RADIOLOGY

A MONTHLY JOURNAL DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES

EDITOR

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Dr. Rollin Howard Stevens

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Rollin Howard Stevens, M.D., 1868-1946[1]

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IN 1952 THE FIRST of a series of lectures on some phase of the radiologic history of medicine was delivered before the Radiological Society of North America by Dr. George W. Holmes, who chose as his subject Dr. Walter Bradford Cannon, the physiologist, to whom gastrointestinal diagnosis owes so much. Two years ago the Historian of the Society, Dr. Howard P. Doub, "lifted up his eyes unto the hills" and added glory to the many accomplishments of Dr. Augustus W. Crane. I am delighted to have been delegated by our President, Dr. Clarence E. Hufford, to contribute the third sketch in this series on my chief, my teacher, and my associate of many years, Dr. Rollin Howard Stevens, the ninth President of the Radiological Society of North America.

Dr. Stevens was born in Canada and was educated at the University of Michigan, with postgraduate work in Germany, Denmark, England, and France. His entire career as a radiologist was spent in Detroit, Mich., where for nearly sixty years he was associated with Grace Hospital. His early background and training, however, made him, in fact, a "citizen of the world," long before President Wilson advocated the League of Nations or Wendell Willkie world citizenship.

It is possible now to look back on Dr. Stevens' full life and analyze his accomplishments, shall we say, from the point of view of the American way of life? This familiar phrase has innumerable meanings and a diversity of interpretations. A better term would be "ways of life"—*American* ways of life—since it has to do with ideals, principles, and beliefs and even includes medical and radiological research.

The true American way of life is concerned with freedom and liberty; more important, it connotes duty, active and vigorous participation in all fields of endeavor. Liberty consists more in duty than in rights. It is to be sincerely hoped that it means, also, that "each and every one of us as individuals must learn to implement that which we have and do for the common good of humanity." If this speculation is even partially correct, Dr. Stevens' life, dutifully dedicated to the science of Radiology, was *his* contribution to the American way of life.

The January 1938 edition of RADIOLOGY, *in toto*, was dedicated to Dr. Stevens, in honor of his attainment of the age of three score and ten, "in full possession of his mental and physical vigor," as Dr. Albert Soiland expressed it in his contributed editorial. I invite everyone, and particularly the members of the Radiological Society of North America, to read this

[1] Presented at the Forty-second Annual Meeting of The Radiological Society of North America, Chicago, Ill., Dec. 2-7, 1956.

edition, especially Percy Brown's fascinating "Chronicle of His Useful Life." Dr. Brown, an intimate friend and for a time an associate of Dr. Stevens, wrote, by way of introduction:

> "One of the most active and vigorous figures in the practice of American radiology today is that of Dr. Rollin Stevens, who now reaches the age of three score and ten prophetically signalized in Scriptural writ, and whose enthusiastic, sedulous, and forthright individuality, crystallized in the service of mankind and polished by the gentle contact of each passing year, continues to reflect from its many facets his faith in his work and in himself in the doing of it, as well as the joy of fulfilment which realization of his life's ideals has assured to him."

That most certainly exemplifies the American way of life!

If time permitted I would read Dr. Brown's tribute in its entirety, as it clarifies the diverse facets of Rollin Stevens' life and interests up to the age of seventy, including civic activity, play, hobbies and relaxation, love of children, and admiration of nature.

His inexhaustible energy and love of nature carried him into the field of mycology and resulted in his becoming an authority on fungi and mushrooms, especially *Amanita*. A new variety of mushroom discovered by him was named in his honor, *Helvella Stevensii*. The gathering and classifying of sea-shells was another hobby, from which he derived a great deal of pleasure. But flowers held a special interest for him, and again he was honored in an allied scientific field by having a variety of Hemerocallis, or day lily, named the Dr. R. H. Stevens.

Because of his understanding of human nature and its associated frailties, Dr. Stevens was instrumental in the organization of what is now known as the Boys' Republic, at Farmington, Mich. His interest in its development and activities for its welfare became an inherent part of his life.

But Dr. Stevens did not retire at the age of seventy. In the last decade of his useful life, he directed prodigious activity to the creation of effective ways and means of combating cancer. All of his energy and vitality were bent in one direction—*fight cancer—prevent cancer*—find more effective and better ways to *treat cancer—save lives—cure cancer!*

It is interesting to study his voluminous plans and his method of strategy to accomplish good for humanity. It could be used now as a model to promote the American way of life in many endeavors. To quote his challenge in 1941 to the Wayne County (Michigan) Medical Society, as Chairman of the Cancer Committee:

> "In Detroit we have a young university, the University of Wayne, which recently received recognition from the Federal Government in that it designated Detroit as a training center for Fellows specializing in Cancer. Why should not Wayne County Medical Society, which is an outstanding unit of its kind in the world, encourage this recognition by promoting, in connection with Wayne University, cancer research and thus bring the eyes of the world to again focus on Detroit as a center of research and service—this time medical—and cancer is its *greatest challenge*."

As Chairman of this Cancer Committee Dr. Stevens drafted plans and proposals for the establishment, equipment, maintenance and function of a Cancer Research Laboratory. Only one came to fruition. His confreres in Detroit know that *his* efforts were mainly responsible for the building of Detroit's Institute of Cancer Research, and he was made its first President.

Through his efforts Drs. Mayne R. Curtis and Wilhelmina F. Dunning, who had worked together in the Crocker Cancer Research Laboratories of Columbia University, New York City, under the direction of Dr. Francis Carter Wood, turned over their colony of rats and their services to the Detroit Institute of Cancer Research. Their work has been endorsed by the American Cancer Society, and many articles concerning their research in genetics and the biology of cancer have been published.

Dr. Stevens relinquished to others much honor and glory which rightfully belong to him, and in so doing exemplified the principles which he advocated in his report to

the Wayne County Medical Society in 1940:

"In this work I hope we all can subjugate any personal prejudice we may have for our own advancement and pull together for a greater institution which some of us may not live to see accomplished, but will enable the younger men and women who follow us to learn more and more about the truth of the cancer problem."

This was Dr. Rollin H. Stevens' American way of life—the way of an uncommon man whose interest was humanity.

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SUMMARIO IN INTERLINGUA

Rollin Howard Stevens, Dr. Med., 1868 a 1948

Iste conferentia historic, presentate ante le Societate Radiologic de Nord-America, honora Dr. Rollin Howard Stevens qui practicava radiologia in Detroit durante quasi sexanta annos. Le autor paga tributo a Dr. Stevens como exemplo del via de vita american per su devotion sin reserva al ideales de servicio. Mention es facite, in particular, de su activitates in le campo del recercas de cancere le quales culminava in le erection del Instituto pro Recercas de Cancere a Detroit.

Current Situation with Regard to Permissible Radiation Exposure Levels[1]

LAURISTON S. TAYLOR[2]

THIS DISCUSSION will deal primarily with permissible radiation levels in relation to occupational exposure. While such levels are intimately related to those for the general population, this phase of the problem will not be discussed in detail.

For the past twenty-five years this country has based its permissible occupational exposure levels on the recommendations of the National Committee on Radiation Protection (NCRP) and the International Commission on Radiological Protection (ICRP). In both cases the exposure levels have been determined primarily by the effect of radiation upon the individual. The current permissible weekly level is based on the premise that a whole-body exposure of 0.3 rem/week for an indefinite period would not produce any detectable harmful effect.

It should be pointed out that at the time these levels were set, in 1948, extensive consideration was given to the effect of radiation on the genetic system and upon the average expected life-span of the occupationally irradiated population. *Handbook 59*, which set forth the recommendations on permissible exposure from external radiation, made numerous references to the fact that this basic requirement might have to be changed in the future (1). This is typified by the following statement:

> "As the applications of atomic energy expand and the number of exposed individuals increases, genetic effects will become more important. Accordingly, it may be expected that at some time in the not too distant future a reappraisal of the situation will become necessary. On the basis of present knowledge of the genetic effects of radiation, it may be predicted that any future revision of permissible doses to the gonads of young persons will be downward. This should be borne in mind, and unnecessary exposure to radiation should be avoided at all times."

Similarly, the effect of radiation on the average life expectancy was considered. The knowledge of a few years ago was vastly less than at present, and even today the question is one in which there is considerable quantitative doubt.

The present permissible exposure levels took into consideration the fact that only a small fraction of the population would be subjected to radiation exposures approaching the maximum permissible levels. No allowance was made, however, for the fact that the great majority of radiation exposures would be considerably below the permissible levels. An additional safety factor was thus automatically introduced. An estimate that there may be as many as 500,000 radiation workers in this country would undoubtedly be on the high side. This would be about one-third of 1 per cent of the population. Considering occupational exposure alone, compared with medical and other sources of irradiation, the total exposure to this group would be regarded as genetically relatively unimportant.

The report of the National Academy of Sciences (2) on the genetic effects of radiation indicated that an exposure of 1,000 r has produced in the radiological profession, a shortening of about five years in the average life expectancy. This figure is probably as good as any that can be attained; yet it must be borne in mind that it is based on statistics including most of the early radiologists, who worked under conditions of almost zero protection. It is believed by many that the estimate of 1,000 r average exposure for these persons is in all probability low and that a more realistic figure may be of the order of 3,000 or 4,000 r. What this exposure actually was, it will never be possible to determine.

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[1] Presented at the Forty-second Annual Meeting of the Radiological Society of North America, Chicago, Ill., Dec. 2–7, 1956, and at the meeting of the American Nuclear Society, Dec. 12, 1956.

[2] Chief, Atomic and Radiation Physics Division, National Bureau of Standards, Washington, D.C.

According to the present permissible levels of 0.3 r per week or 15 r per year, an x-ray worker could receive as much as 600 r in forty years of working life-time, or about 300 r up to the end of his genetically important period. There can be little argument that in the light of present knowledge this amount of exposure may be unreasonably high; yet, as far as the individual is concerned, there is no evidence at present that exposures at these rates have caused detectable injury. In fact, there is no evidence that the higher levels of 100 r per year from 1928 to 1936, or 36 r per year from 1936 to 1947, have resulted in detectable damage. It should, of course, be pointed out that average shortening of life expectancy must be regarded essentially as an undetermined injury.

Even with the low levels of 0.3 rem per week in use since 1947, most atomic energy plants have found that they could operate satisfactorily at levels ranging from one-third to one-tenth of this value without seriously interfering with their operations. It is erring on the safe side, to say that in no large atomic energy plant in this country has the average exposure per monitored individual exceeded, or in most cases even approached, 0.1 rem per week (3).

It should also be pointed out that, when one considers the problem of radiation effect on life-span, one thinks of whole-body exposure. In fact, a substantial fraction of the persons at present receiving occupational exposures in the medical field have only small portions of the body exposed. Under such circumstances, the effect on average life expectancy is very much less.

The National Committee on Radiation Protection in 1946, and the International Commission on Radiation Protection in 1950, undertook extensive studies of the whole question of permissible exposure, and it was during this period that the present levels were set. In 1952 there was a special meeting of the ICRP, in conjunction with the Joint Committee on Radiobiology of the International Union of Pure and Applied Physics, for the specific purpose of studying the genetic effects of radiation exposure. Participating in this were a number of geneticists from several countries. The basic problem before the Commission at that time was consideration of an average population dose to the gonads, during the reproductive period. The acceptable values then ranged from 3 to 20 rem over and above the dose due to natural and background radiation. (These bracket the figure suggested by the report of the National Academy of Sciences in June 1956.) At the time, it was considered by the ICRP that the fraction of the population receiving radiation exposure was so small that immediate concern over the total population dose was not necessary; on the other hand, there has been and still is clear recognition of the fact that this situation will almost certainly change as the use of radiation increases throughout the world.

At meetings of the ICRP in Geneva in April 1956, the genetic problem was again considered, and a figure of about 10 rem of man-made radiation was recommended as a reasonable average per-capita dose for the future. It should be emphasized that with our present practices it is not likely that the increase of occupational exposure will contribute very significantly to this total exposure level.

It has been decided by the NCRP, on the basis of present evidence, that the most important new limitation should be one relative to gonadal exposure to individuals during the reproductive period. In this respect it was considered that for occupational exposure a dose of about 100 rem up to age forty would not involve an unacceptable risk. Similarly an exposure of up to 250 rem by age seventy would not be regarded as unacceptable. The influence of exposure received beyond the reproductive age would in the main be through its effect on expected life-span. Operationally, it would seem to be just about as easy to continue, beyond age forty, the same permissible levels allowed below that age.

In these considerations it is clear that

the emphasis has shifted from the exposure of the individual to exposure of the population. At the same time it has to some extent shifted from a weekly or monthly exposure level to the exposure accumulated over a lifetime.

In principle, permissible exposures such as indicated above may be considered as adequate for all operational and regulatory purposes. Unfortunately, in practice this is not feasible and, as a result, it has been necessary to develop a series of derived permissible exposure levels over varying periods of time, and in relation to the age of the individual.

The difficulty in dealing with a single number giving the individual's total permissible life-time exposure, is introduced because of the fact that the safety *recommendations* of a few years ago have now entered our legal machinery and become *legal requirements*, backed up by varying degrees of enforcement. For the twenty-five years beginning with the appearance of the first NCRP *Handbook* in 1931, radiation safety control in this country was on the basis of recommendations backed up by voluntary compliance and good sense. It must be admitted that there were times when neither of these prevailed, but in the great majority of situations they did. A substantial number of legal cases involving over-exposure to radiation, were settled one way or the other on the basis of NCRP *Handbooks* without any of these ever being legally adopted in the form of codes or laws.

The basic scientific aims and information regarding permissible radiation exposures have been well understood for a number of years. On the other hand, there have been situations where the information was capable of misinterpretation by persons inexperienced in the field. Partly as a result of this, there has been an increased tendency on the part of industry and state and federal governments to call for strict codification and the development of radiation protection rules, wherein a situation becomes either black or white. Here begins our trouble.

Let us consider as acceptable a total accumulated exposure of 250 rem over a period of fifty years, up to age seventy. It is obviously impossible, with any means now known, to measure and record this exposure for each individual. Therefore, the operator might decide to make his measurements over a period of ten years, assuring that no employee would receive more than 50 rem over that period. This is still impractical, because we have no instrument that can be worn for ten years to record the dose received. Furthermore, an occasional careless employer may allow an individual to receive his ten-year dose of 50 rem in three years and, having done this, simply let the man go because he is no longer useful in radiation work.

For purposes of design and operation, one might want to integrate the exposure over a shorter period of time, say one year, in which case the average allowable amount would be 5 rem. This is also difficult to control, but it is suspected that, before too long, adequate instrumentation will be obtainable so that this can be done within adequate accuracy limits. At least as far as radiation operations are concerned, the level of 5 r per year is a much better figure to work with.

The importance of limiting exposure during the child-bearing age has been mentioned. For this purpose, the NCRP is recommending a permissible occupational exposure of approximately 60 rem up to age thirty, and an additional 50 rem up to age forty. Here again it is important to assure an adequate distribution of dose over time. If one considers that 50 per cent of the children are born to parents by the time they reach age thirty, and 90 per cent by the time they reach age forty, it is clearly more important to curtail exposure at the lower end of this age range than at the upper end. There would be a large difference between the total genetic damage due to an exposure of 50 rem at age twenty and the same exposure at age thirty. At the age of forty this exposure would affect only 10

per cent of the genes that might be expected to be transmitted. Consequently, there is some wisdom in stating that, for operational purposes, the occupational exposure of individuals should not exceed an average of 5 rem per year.

But even here there are difficulties. Suppose that the NCRP were to recommend 5 rem per year for the basic permissible level as compared with the present level of 0.3 rem per week. As soon as this figure is even mentioned it will find its way into various laws and regulations. Everything automatically becomes black or white, and any individual who may be so unfortunate as to receive 5.001 rem in any one year might think he had legal grounds for seeking redress from his employer. This is, of course, sheer nonsense; yet it is difficult to develop a law in which there is adequate latitude for reasonableness in this respect.

It has been suggested that the yearly limit be specified as 5 rem plus or minus 20 per cent. This would certainly simplify planning and operations, but it is very uncertain as to how and whether this could be dealt with to the satisfaction of our legal authorities. Another suggestion has been to specify a total exposure not exceeding 50 rem in ten years delivered at an average rate not exceeding 5 rem per year. This would be unacceptable because one should not allow an individual at the beginning of his reproductive age to receive so large a dose. If he is to have a large dose, it is much better that he receive it after the conception of his last child; and time of conception, of course, changes with age.

Still another alternative is to specify a 5 rem per year average but with the further stipulation that no one be allowed to receive in excess of 10 rem in any consecutive two years. Even this would allow some abuse, but such a provision would simplify many operations. For example, if an individual received, say, 6 rem in one year, it would be necessary to insure that he could receive no more than 4 rem in the following year. If one decides on an average figure of 5 rem per year the plant will be designed accordingly. The designer will possibly go further than this, since control over a year is not always easy. He may well, for purposes of simplicity, make his designs such as not to allow the exposures to exceed 0.1 rem per week. (This is one-third of the present maximum permissible exposure.)

All of the discussion thus far leads to one almost unescapable conclusion—namely that the maximum permissible exposure levels are going to be reduced to one-third of what they are at the present time. One could then ask why all the fuss about the various exposure levels that have been discussed above? Except for the exposure limitation for genetic reasons in the twenty- to forty-year bracket, all of the levels are so low as to make virtually impossible any distinction between a dose delivered over a few months and the same dose delivered over a few years.

One could well ask, then why not accept the basic figure of 250 rem up to age seventy and leave the rest of it up to management—or even do the same for 50 rem per ten years? The difficulty, however, is that various individuals will interpret the limits in various ways and, unless the permissible dose is integrated over a relatively short period, a given employer might use up all of the employee's permissible exposure, making it impossible for the employee to shift positions or to continue work in his chosen field. It must be borne in mind, however, that the permissible levels that are expressed in terms of a week, thirteen weeks, a year, or even ten years, are regarded as numbers to be used for design and operational purposes. There is nothing sacred about any of them, and yet failure to introduce such time-dose limitations will encourage such a high degree of non-uniformity of permissible exposure in the radiation field as to make employment in radiation work unattractive.

Where an installation or individual can provide assurance that the monitoring and recording methods are adequate, the

widest latitude should be allowed in the use of permissible exposure levels with the employees. It is not known how this can be worked out legally, but it is fairly certain that, in operations of any substantial size, it would pay the employer to have adequate measuring and recording systems and then be allowed wide latitude in his use of any of the various permissible levels mentioned above.

The main difficulty may occur with the small users of radiation sources, where there is transient employment and the cost of monitoring equipment may be incommensurate with the cost of the operation. For employees, the permissible exposure levels integrated over one week, or at the most three months, would in all probability be the most feasible.

As a solution to many of the problems outlined above, the National Committee on Radiation Protection has recently revised its earlier recommendations on the maximum permissible exposures to radiation (4). The basic figure of 0.3 rem/week, together with the penalty allowance of 3.0 rem/13 weeks, at present in use, will be continued. So also will be the allowance of 15 rem/year (allowing for two weeks vacation). These figures will, however, be subject to a further basic limitation to insure that the dose is not accumulated too rapidly, or reaches totals per individual that are not considered acceptable. If we allow an accumulated dose, over fifty years, of about 250 rem up to age seventy, this would amount to an average of about 5 rem/year.

Most of the operational problems can be solved by means of a simple formulation that implies the building up of a bank of reserve exposure that may be called upon as needed. The new requirement will be that, at a given age of 18 or over, an individual may be allowed to accept a dose such that his total accumulation in rem will not be in excess of 5 times the number of years of age over 18. Thus, at any time, exposure must be less than $5\ (N-18)$ rem where N represents the person's age. This automatically holds down exposure at the younger ages, where it is most critical. It gives leeway at the older ages, where it is less critical. Thus if a person starting work at age eighteen receives only 1 rem per year for five years, his total will be 5 rem as compared with 25 rem permitted. He has thus accumulated a reserve of 20 rem. Should occasion then demand a larger dose, he can take up to 15 rem in the next year and still have a reserve of 10 rem. If a person starts radiation work at a later age, he will have automatically built up a reserve and can take larger annual doses—so long as the accumulation remains less than the age pro-rated maximum. This procedure eliminates the need for the present rule, allowing larger exposures for persons over forty-five.

For design or planning or operational purposes, it may be desirable to use average weekly, monthly or quarterly levels and do the monitoring accordingly. Under the new rule this will be permitted. Where it is desirable to keep monitoring and recording procedures to a minimum, it would probably be wise, in many cases, to operate on a weekly or monthly basis. The choice of procedure will depend largely upon the economics of any particular situation.

When a person's exposure in prior employment may be unknown or undocumented, it will be presumed to have been the maximum permitted up to his particular age.

It might be pointed out that the above levels need not be modified by the acceptance of one emergency exposure of 25 rem during a person's lifetime. Also it might be noted that these exposure levels are not modified by any radiation exposure received for medical reasons. On the other hand, it would be prudent for the employer to take any especially large medical exposures into consideration in the assignment of an individual to radiation work.

The rules given in *Handbook 59* will be continued, but some of them will be modified by provisos related to a yearly

limitation of 5 rem to the blood-forming organs, gonads, and lenses of the eyes, and to a limitation of 10 rem to the skin.

Permissible radiation levels for internal emitters will conform to the general principles already outlined. Where the critical organ is the gonads or the whole body, the maximum permissible concentrations for air and water will be one-third the present values specified for radiation workers. This will probably apply to less than half a dozen radioisotopes. Where single organs are regarded as the critical organ, the presently accepted concentrations may be continued. For persons living in the neighborhood of controlled areas, the maximum permissible concentrations should be further reduced by a factor of 10.

Atomic and Radiation Physics Division
National Bureau of Standards
Washington 25, D. C.

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SUMMARIO IN INTERLINGUA

Le Situation Actual in Re Nivellos Permissibile de Radio-Exposition

Iste articulo se occupa primarimente con le nivellos permissibile de radiation in exposition professional. Viste le nunc acceptate nivello permissible de 0,3 rem per septimana o de 15 rem per anno, un laborante roentgenographic pote reciper usque a 600 r in 40 annos de vita active o circa 300 r usque al fin de su geneticamente significative periodo.

In le passato recente on nota le tendentia de sublinear le signification del dosage accumulate a longe vista, con attention special prestate al exposition gonadic e al effectos genetic. Le Commission National pro le Protection Contra le Radiation ha fixate circa 100 rem usque al etate de 40 annos como non representante un risco inacceptabile, plus 250 rem additional pro le periodo usque al etate de 70 annos. Su recommendation specific es 60 rem usque al etate de 30 annos e 50 rem additional usque al etate de 40 annos.

Es discutite le problemas que inhere in le effortio a establir un valor definite pro le exposition in le curso del vita total. Varie possibilitates pro solver ille problemas es considerate. Il pare ineluctabile concluder que le nunc acceptate maximos permissibile de exposition va esser reducite in le futuro per circa duo tertios. Al presente, le cifras fundamental de 0,3 rem per septimana e de 15 rem per anno va esser mantenite, con le possibilitate, il es ver, de limitar los additionalmente pro assecurar que le dose non se accumula troppo rapidemente o attinge un total que es inacceptabile. Altere modificationes va concerner le exposition de certe tessutos, per exemplo del organos hematopoietic, del gonades, del lentes ocular, e del pelle.

Delayed Effects of Ionizing Radiation[1]

CHARLES E. DUNLAP, M.D.

PRACTICALLY all the known biologic changes produced by ionizing radiation are in a strict sense delayed effects, since they develop only after a significant latent period. It is customary, however, to limit the term "delayed effects" to those injuries that first become apparent several months or years after exposure. At times no sharp dividing line separates the early from the delayed reactions, but as a rule the two are easily distinguished. Tissues in which delayed effects are destined to appear seldom show a steady progression of injury from the early to the late reaction. Characteristically, the acute response subsides and the tissues resume a fairly normal appearance or at least reach a stable state of relative ischemia. At any time thereafter the late lesions may develop.

Most delayed radiation injuries are the consequence of accidental or misguided overexposure. They may develop following large therapeutic doses given in single or multiple exposures or as the cumulative effect of small continuous or intermittent doses absorbed accidentally over the course of years. All types of ionizing radiation, whether delivered from internal or external sources, are effective provided the doses are sufficient. No tissue of the body is immune, but in practice the majority of the late lesions are found to fall into a limited number of well recognized types.

Radiation injuries may be separated into two major categories, according to whether they are local or general in nature. Localized lesions usually result from local irradiation but some local lesions, such as cataract, may also follow whole-body exposure. In like fashion, generalized effects are seen most often when either a large area or the entire body has been exposed, though heavy local treatment at times produces systemic effects.

No detailed description of the various delayed lesions will be attempted, but a few of the commoner types deserve brief mention. Late radiation ulcers usually result from localized radiation therapy. They occur most often in the skin but are seen also in the rectum and bladder and occasionally elsewhere. Even though the dose has been of such a size that late lesions will ultimately develop, the early acute reaction usually heals, leaving a poorly vascularized tissue which is often the seat of telangiectases, chronic edema, and fibrosis. Ulceration begins after an indeterminate latent period of months or years and extends progressively to produce a sharply punched-out ulcer crater with a brawny, indurated, ischemic base. Once the ulcer has developed, it heals poorly if at all, and even if it heals is prone to recur. Ischemia appears to be a major underlying factor in the pathogenesis of ulcerating lesions, although the onset is often precipitated by some secondary injury.

Chronic radiation dermatitis, resembling in many respects spontaneous senile degeneration of the skin, may persist for many years without ulceration. Such skin, whether or not it ulcerates, shows a striking predisposition to the development of keratoses and carcinoma at any time from two to thirty years or more after the beginning of exposure. Severe radiation dermatitis and its sequelae may result from ill-advised radiation therapy administered to any region of the body. It is seen most often as an occupational disorder, involving the dorsum of the hands and fingers of older radiologists who, in their earlier years of practice, failed to observe proper protective measures.

[1] From the Department of Pathology, School of Medicine, Tulane University, New Orleans, La. Presented in part at the Forty-second Annual Meeting of the Radiological Society of North America, Chicago, Ill., Dec. 2–7, 1956.

Heavily irradiated bone undergoes aseptic necrosis without necessarily producing symptoms. Pathological fracture may result and, if secondary bacterial infection occurs, an intractable and often painful osteomyelitis is the usual result. Radiation necrosis of bone with associated imperfect attempts at repair is customarily referred to as "radiation osteitis," although the term leaves something to be desired. It may result from heavy external irradiation or from osseous deposits of radium, strontium 90, or other radioactive substances. Osteogenic sarcoma often develops in regions of radiation osteitis and can be produced with considerable regularity by radioactive substances deposited in the bones of experimental animals or human beings. Less commonly, bone sarcoma may follow external roentgen irradiation. Mature cartilage withstands radiation fairly well, but in young subjects the growing epiphyseal cartilages are easily damaged, with resulting early epiphyseal closure and permanent deformity of the skeleton.

The kidney is relatively radioresistant, and fairly large local doses may be applied with little evidence of injury. Full tumor doses to the kidney, however, may bring about vascular changes and hypertension similar in many respects to spontaneous nephrosclerosis. There is also evidence that general body irradiation in amounts that expose the kidney itself to much smaller doses will accelerate the development of nephrosclerosis.

General body irradiation, like heavy local exposures, may bring about permanent damage to the bone marrow, with sustained or even progressive leukopenia and anemia. Such myelophthisic states respond poorly to all known forms of treatment. Inadequately protected or careless professional radiologists often show a mild leukopenia with relative lymphocytosis, and occasionally a sustained or periodic absolute leukocytosis develops. This is believed to represent an excessive compensatory response to repeated minor injuries. Irradiated subjects, both men and animals, show an increased incidence of leukemia which is clinically indistinguishable from spontaneous leukemia.

One of the most puzzling and interesting of the delayed responses to radiation, which has only recently become firmly established, is a non-selective overall acceleration of the many changes associated with the natural aging process. Accompanying the evidences of premature aging there is a demonstrable shortening of the life span not attributable to any particular type of disease but rather to a general increase in the same disorders that ordinarily increase with age. The ability of radiation to accelerate aging and shorten the life span has been demonstrated chiefly in experimental animals, but evidence is accumulating that man is probably subject to the same effects.

It is generally agreed that radiation plays some part in causing all these and many other disorders, yet it is hard to account for such an array of dissimilar changes on the basis of a single form of injury and a common chain of pathogenesis. Two of the most striking changes seen in irradiated tissues are death of cells and damage to blood vessels. These two changes are often important factors in the production of lesions. In acute reactions, massive cell death is easy to demonstrate, particularly in radiosensitive tissues such as the bone marrow, lymph nodes, and intestinal mucosa. The rate at which cells are dying reaches a peak within a few days after exposure and then tapers off in a slow decrescendo during the succeeding weeks. The killing of cells is such a dramatic event and also such a fundamental factor in successful radiotherapy that its general role in the pathogenesis of radiation reactions has been overemphasized.

Late radiation lesions develop in fairly well stabilized tissues, in which cell death is probably less important than sublethal cellular injuries. The cell population that makes up an irradiated tissue includes all cells, regardless of functional damage, that have managed to retain the minimal

metabolic machinery necessary for individual survival. It is little wonder that such tissues recover poorly after any injury and show a diminished ability to withstand all sorts of secondary insults such as exposure to sunlight or additional ionizing radiation, extremes of heat and cold, ischemia, chemical irritation, and bacterial infection. Impaired blood supply is often important in predisposing these tissues to injury, but something more than ischemia is necessary to explain the onset and the characteristics of many of the delayed effects.

The concept of cell injury short of death is basic to all studies in radiation genetics and is useful as well in understanding somatic injuries. The various special functions of a single cell are not equally vulnerable to injury, and it can be shown that graded doses of radiation will suppress successive functions without killing the cell. As early as 1911, Hertwig (1) reported a provocative series of experiments on the fertilization of normal frog eggs with irradiated sperm. Small doses resulted in sundry abnormalities in the embryos; larger doses were followed by more serious malformations, with early death of most of the embryos. However, a slight further increase in dose appeared to diminish the severity of injury and many embryos were apparently normal. Hertwig suggested that at the two lower dose levels, injury to the chromosomes of the sperm explained the sublethal or lethal abnormalities in the embryos. At the higher dosage the spermatozoa retained their special functions of motility and ability to penetrate the cell membrane of the ovum, but the male chromosomes were so badly damaged that they took no further part in the process and the resulting embryos were parthenogenetic haploids derived solely from the ovum. Among the more familiar radiation injuries to cell function in mammals one might mention inhibition of ciliary activity of bronchial epithelium, suppression of secretion of salivary and gastric glands, partial suppression of phagocytosis in tissue macrophages, impairment of antibody production in reticuloendothelial cells, suppression of function in thyroid cells, and delay in the formation of granulation tissue and epithelization in healing skin wounds. Some of these injuries are reversible, but others are permanent and apparently transmissible to subsequent cell generations.

The loss of specialized cell functions may decrease the ability of tissues to withstand unrelated forms of injury. The importance of secondary agents in the pathogenesis of late lesions appears most clearly in cases of post-irradiation bacterial infection. Here systemic results of previous irradiation, such as granulopenia and diminished antibody production, combine with diminished local tissue resistance to favor bacterial invasion and growth. Radiation osteomyelitis has already been mentioned; a less familiar example is furnished by radiation ulcers of the intestine. Friedman and Warren (2) in an instructive series of experiments demonstrated that irradiation of the intestine predisposes to ulceration but does not of itself determine the size, shape, location, or even the occurrence of the lesions. Ulcers did not develop in all animals subjected to the same treatment, and such ulcers as did develop were not selectively located in sites that had received the largest doses. On the contrary, the ulcers were distributed in random fashion anywhere along the loop of irradiated intestine. The factors believed responsible for initiating and localizing the ulcers were incidental mechanical trauma and secondary bacterial infection of the partially devitalized intestinal wall.

In some of the delayed reactions in which secondary precipitating causes have not been identified, it is possible that radiation injury is the only causal factor. However, in the long list of late effects attributed to radiation, it is interesting to note the virtual absence of highly specific, unique or unfamiliar lesions. Biological systems, in spite of their complexity, are capable of only a limited number of

basic responses to injury, and it is perhaps not surprising that an acute radiation dermatitis, for example, bears many similarities to sunburn or thermal burn. Acute inflammatory reactions in general do not tend to be highly specific. In response to certain injurious agents, however, whether bacterial, viral, parasitic or chemical, tissues often show reaction patterns so distinctive that the causative agent or disease can be identified on this basis alone. Although ionization certainly plays a major part in initiating many of the ischemic, atrophic, degenerative, fibrotic, necrotic, dysplastic, metaplastic, and neoplastic changes associated with various radiation injuries (3), I suspect that comparatively few fully developed late radiation lesions are caused exclusively and specifically by radiation. When the lesion in question is also known to occur in essentially identical form in non-irradiated subjects, there is additional reason to suspect the intervention of secondary etiological factors.

Radiation leukemia will serve nicely to illustrate some of the points I have tried to make concerning delayed effects in general. Although cell death and impaired circulation are important features in many radiation reactions, they play no obvious part in producing leukemia. The effective doses are often too small to result in detectable vascular injury, and leukemia does not develop until long after the phase of cell death has subsided. If we adopt the conventional assumption that leukemic cells are the progeny of surviving hematopoietic cells which have been damaged genetically or metabolically by prior irradiation, then cell death would not enter the picture.

Although the cause of human leukemia is unknown, there is evidence that exposure to radiation can play a part in its pathogenesis. The incidence of leukemia among professional radiologists, while not high in absolute terms, is some eight to ten times higher than among physicians in general. Patients subjected to therapeutic irradiation for ankylosing spondylitis also show an increased incidence of leukemia, and evidence is accumulating that in children who have been exposed to small doses *in utero* or those subjected to thymic irradiation after birth leukemia is somewhat more likely to develop than in their siblings. The increase in leukemia among survivors of the Nagasaki and Hiroshima bombings has shown a good correlation with the dose of radiation as estimated from the victim's distance from the center of the blast. Experiments with rats and mice confirm the scanty human data at every point and indicate that the incidence of leukemia can be significantly increased by long-term whole-body irradiation at low intensities as well as by fractionated exposures within the therapeutic range and by single massive whole body exposures. In the face of all this evidence, it is tempting to assume that a direct cause and effect relationship exists between radiation and leukemia. Other facts, however, suggest that the relationship may be indirect. Among these are the occurrence of leukemia in unirradiated subjects, the long and unpredictable time lag between exposure and the onset of leukemia, the fact that the disease develops in only an occasional person in an irradiated group, and the discovery that filterable agents are capable of inducing leukemia in rats, mice, and fowl.

A hypothetical series of experiments may further illustrate how difficult it is to prove a direct causal relationship between radiation and the lesions attributed to it. Rats are known to be susceptible to endemic typhus fever, but the natural disease is often relatively mild and many rats survive the infection. In 1932 Zinsser and Castañeda (4) found that in rats given whole-body irradiation before experimental infection a fulminant and rapidly fatal form of the disease developed. Now let us assume that the cause of typhus is not known and rats are being irradiated for some reason in a region where there is a natural reservoir of typhus and where the intermediate carriers, rat fleas and rat

lice, are present in the animal quarters. Under these conditions, irradiated rats would come down with a violent disease characterized by fever and scrotal swelling. At autopsy, gross and microscopic lesions would be found similar to those seen in human typhus. The disease might not affect all irradiated animals nor would it develop at a fixed time following irradiation, since its onset would be determined by the accident of infection. However, no similar disease would develop in unirradiated rats. These findings would naturally suggest that radiation could be the cause of endemic typhus. A careful investigator, however, would want additional proof, so the experiment might be repeated a number of times on rats and then tried on several groups of mice. Each experiment would yield essentially identical results. The investigator now feels on firmer ground but, in the meantime, an article has appeared in a foreign journal proposing that endemic typhus is actually caused by *Rickettsia prowazekii*. Samples of this organism are therefore obtained and injected into normal rats. In many of the rats fever develops, but few deaths occur. In irradiated rats, on the other hand, the rapidly fatal disease develops that the investigator has learned to recognize as rat typhus, regardless of whether the animals have been injected with rickettsiae. A survey of human typhus is then launched and, to the investigator's delight, it reveals a statistically significant increase in the incidence of endemic typhus not only among radiologists but also among patients who have received therapeutic radiation. One small but troublesome difficulty remains. Some cases of typhus occur in people who have never been irradiated. However, it is common knowledge that everyone is subjected to an unremitting, lifelong bombardment by low-intensity background radiation and this could reasonably account for the sporadic cases. The chain of scientific evidence is now complete, proving that ionizing radiation is the direct cause of endemic typhus and an increase in malpractice insurance rates promptly follows.

This imaginary series of observations parallels in certain respects the present evidence relating radiation and leukemia. It is not presented in support of the virus theory or any other notion of the etiology of cancer, but only to emphasize the need of caution in concluding that radiation injury is the proximate cause of disorders that develop subsequent to irradiation.

A variety of disorders have been found to occur with increased frequency following irradiation. Those that first appear some months or years following exposure are known collectively as delayed radiation effects. In some instances the causative role of radiation is obvious, as when a chronic skin ulcer develops in the precise area where an excessive dose of radiation has produced severe local injury to the skin. Other delayed effects, such as premature aging and leukemia, are clinically indistinguishable from the corresponding spontaneous disorders and statistical evidence is necessary to demonstrate a relationship to prior irradiation. It is clear that radiation may play either a dominant or a minor role in the pathogenesis of different delayed effects and the responsibility that radiation bears for the ultimate consequences will vary accordingly.

In order to indicate these differences, an attempt should be made to differentiate those lesions that are the direct and predictable result of a given exposure to radiation, those in which important secondary factors such as bacterial infection must be combined with radiation injury to produce the final lesion, and those in which radiation is involved only as a remote, nonspecific, predisposing influence. It is scientifically important to recognize that there are these different categories of delayed radiation injury, and from the practical aspect such differences may be equally significant. Radiologists are held accountable for any untoward results of

the treatment they administer, and the law makes important distinctions between the proximate cause of an injury and a remote cause. Perhaps the greatest value of recognizing that factors other than radiation are often the precipitating causes of delayed injuries is the possibility that the lesions themselves may be prevented by recognizing and controlling these precipitating factors.

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SUMMARIO IN INTERLINGUA

Effectos Tardive del Radiation Ionisante

Le majoritate del tardive lesiones radiational es le consequentia de hyperexposition per accidente o error. Illos es de duo typos principal: (1) Lesiones localisate, usualmente como resultato de irradiation local, e (2) lesiones generalisate, plus communmente post irradiation de un grande area del corpore o mesmo del corpore total sed a vices etiam post intense exposition local.

Le plus commun tardive lesiones de radiation include ulceres radiational que usualmente affice le pelle sed que se trova etiam in le recto e le vesica, chronic dermatitis radiational, aseptic necrosis ossee, sarcoma ossee, insultos reno-vascular que in multe respectos resimila nephrosclerosis spontanee, e alterationes del medulla ossee con augmentate incidentia de leucemia. A istos on pote adder le plus recentemente establite responsa del non-selective acceleration general de multe alterationes characteristic del normal processo invetulatori.

In certe casos le rolo causative de radiation es obvie. In alteres, corroboration statistic es necessari pro demonstrar un connexion con irradiation anterior. Il es clar que radiation pote haber un rolo dominante o subordinate. On deberea effortiar se a differentiar inter (1) lesiones que es le directe e predicibile resultato de un date grado e typo de exposition, (2) lesiones que occurre solmente si importante factores secundari es combinate con le radiation, e (3) lesiones in que radiation entra solmente como remote e non-specific influentia predisponente. Forsan le plus grande valor de recognoscer que factores altere que radiation es le causas precipitatori de lesiones tardive resta in le possibilitate de prevenir le lesiones per regular ille altere factores.

Genetic Considerations in Establishing Maximum Radiation Doses[1]

JAMES F. CROW

SINCE MULLER'S experiments thirty years ago, it has been known that ionizing radiations increase the mutation rate and therefore are to some extent a genetic risk. Recently interest in this question has been stimulated by the realization that wide use of nuclear power may lead to a substantial increase in population exposure. A further factor that has increased the concern of geneticists has been the finding that mice are about 15 times as sensitive to radiation induction of mutations as *Drosophila*, which had formerly served as the sole basis for inferring human risks. It is likely that doses too small to cause any noticeable somatic damage may have appreciable genetic consequences; hence genetic considerations are primary in setting maximum dosages for the total population.

The essential difficulty in setting dose limits is: Although there is general agreement that any increase in radiation exposure is genetically harmful, it is not possible to say with any assurance *how* harmful. This uncertainty prompted the British committee (1) to say: "Nowhere in our report have we been more conscious of the difficulties of the task which we have undertaken, and of the limitations of the knowledge at our disposal, than in considering the genetic effects of radiation." The American report (2) includes similar statements. But despite this uncertainty, the two reports, prepared independently and approaching the problem from different directions, reach remarkably similar conclusions regarding maximum permissible exposures. The American report recommends that all humanly controllable sources of ionizing radiations be so restricted that the gonad dose from conception to age thirty does not average more than 10 r, in addition to background. The British report does not set a definite figure, but says, "Those responsible for authorizing the development and use of sources of ionizing radiation should be advised that the upper limit, which future knowledge may set to the total dose of extra radiation which may be received by the population as a whole, is not likely to be more than twice the dose which is already received from the natural background; the recommended figure may indeed be appreciably lower than this." With the present background estimates of 3 to 5 r, this is practically equivalent to the 10 r of the American report.

My intention is to summarize briefly the present state of knowledge of the genetic effects of radiation in man. In particular I wish to indicate the degree of uncertainty of various conclusions, the assumptions that must be made, and the extent to which it is necessary to depend on data from experimental animals.

SOME GENERAL GENETIC PRINCIPLES

Three well established genetic principles are prerequisites for a consideration of the problem of radiation effects in man.

1. Mutations occur spontaneously throughout the body, presumably in all kinds of cells. From the standpoint of heredity, however, those occurring in somatic tissues or at post-reproductive ages need not be considered. The mutations of importance for future generations are those that take place in the germ cells between conception and reproduction.

2. Almost all mutations that have effects sufficient to be detected are harmful. This is to be expected on theoretical grounds but it is also a matter of direct observation in experimental animals and

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in man. This rule also appears to apply to mutations causing minor or invisible effects. For in *Drosophila*, where special technics and large populations are available, it has been found that mutants causing embryonic or larval death are much more common than those causing obvious abnormality; still more frequent are those leading to a small statistical impairment in the survival rate. On the basis of this experience with fruit flies, we should expect that the most frequent human mutations would not cause gross physical abnormalities, but minor impairments of body functions leading to increased susceptibility to disease, lower life expectancy, reduced fertility, etc.

3. Deleterious mutant genes are eventually eliminated from the population, since they decrease the viability or fertility of the individuals carrying them. In the long run, a mutant causing only a minor impairment can do the same amount of harm as a more serious one, since it is not so quickly eliminated and therefore affects a larger number of individuals. The effect of an improvement in the environment (such as a medical advance) will usually be to ameliorate, but not remove entirely, the deleterious effect of a mutant gene. The harmful effect is diluted and spread over a larger number of generations, but on the average the increased number of persons affected compensates for the lesser effect on each, so that the total harm remains about the same.

There are also two principles of radiation mutagenesis relevant to this discussion.

1. Radiation increases the mutation rate. The evidence for this statement from experimental animals and plants is, of course, overwhelming. There are also some human data. The studies on the children of the Hiroshima and Nagasaki survivors are inconclusive, but two other studies are suggestive. Macht and Lawrence (3) found from questionnaires that stillbirths, neonatal deaths, and congenital abnormalities were more frequent (19.8 per cent) in the children of radiologists than in the controls (17.3 per cent), (rates adjusted for parity differences). This difference is significant by the usual statistical criteria, but one cannot exclude the possibility of biases likely to be encountered in any questionnaire study. Turpin (4) reported sex-ratio changes in the children of parents who had received heavy therapeutic pelvic irradiation. Recessive X-chromosome mutations would be expected to affect males more than females, since in the latter they would be "covered" by their normal alleles. Since the male gets his X-chromosome from his mother, an increase in frequency of sex-linked mutations would appear as a decrease in the proportion of sons from irradiated mothers. Dominant X-chromosome mutants would have a contrary effect, for males transmit an X-chromosome only to their daughters. The data of Turpin show a statistically significant deviation in this direction, *i.e.*, a larger proportion of male children when the father is irradiated than when the mother is. However, this study also is based on questionnaires.

2. The number of mutations produced is directly proportional to the dose in roentgens. The linear proportionality over wide dose ranges has been shown in several organisms, especially in *Drosophilia*. There are departures from linearity at high doses—too high, indeed, for consideration in evaluating the human problem. A major assumption of geneticists is that the linearity extends to lower doses. Experimental verification in *Drosophila* has been carried to as low as 25 r, but at such dosages the mutation rate is so low as to be seriously confounded with the background spontaneous rate. However, if the mutation frequency from higher doses is extrapolated backward linearly to zero dose, a value is obtained within the experimental error of the spontaneous rate. Furthermore, from purely physical considerations, at such low doses the likelihood of coincidences of ionizations of independent origin is very slight. There is good reason, therefore, to

believe that the direct proportionality continues down to zero dose.

The proportionality between dose and mutation production holds irrespective of intensity or spacing, *i.e.*, a total of 100 r has the same effect if delivered in one minute as if given at the rate of 1 r per minute or in sub-units spaced some time apart. There are numerous exceptions to this rule, but not, I believe, of such consequence as to affect seriously the general principle. For example, mature spermatozoa are about four times as susceptible to radiation-induced mutation as earlier stages. But, relative to the entire life span, genetic effects of mature sperm irradiation must account for a very small (and often preventable) part of the total effect. Another complication is due to ion-density effects, but the differences are not great (*e.g.*, neutrons are a little less than twice as effective as x-rays for mature sperm in *Drosophila*), and with the kinds of ionizing radiation most frequently encountered the assumption of the RBE of 1 is a satisfactory approximation.

Finally, gross chromosomal changes violate all these rules, in that they show a non-linear response, dose intensity effects, and strong effects of ion density differences. But these gross changes are probably of little consequence in the assessment of human radiation damage, for three reasons: Most gross aberrations induced prior to gametogenesis lead to the death of the cell before it develops into a sperm or egg and hence the effect is not transmitted. Secondly, gross changes that are transmitted usually cause death of the embryo at a very early stage, which in the human population means that the death would usually be undetected and relatively unimportant. Thirdly, at the low doses likely to be encountered by man, "point" mutations would be expected to predominate over complex chromosome changes.

By combining the third genetic principle with the second radiation principle, geneticists conclude that: *The genetic harm to a population, however felt and however measured, is roughly proportional to the total radiation received between conception and reproduction.* This is true irrespective of how or at what intensity the radiation is received, or how it is distributed among the members of the population; if 1 per cent of the population receives 100 r the effect is the same as if all had received 1 r.

QUANTITATIVE ESTIMATES

The conclusions of the previous section imply that there is no such thing as a "safe" dose. Any increase in radiation, however small, involves a risk proportional to that amount. It is manifestly impossible to eliminate man-made radiation entirely, so it becomes a matter of balancing opposing factors—the genetic risk against the beneficial effects of radiations in industry and medicine.

Thus, it is necessary to attempt some sort of quantitative assessment of radiation damage. For this we must rely almost entirely on experimental animals, as the human data provide practically no quantitative information. I shall use human data where available, mouse data when no human information exists, and if neither mice nor men are informative I shall reluctantly call on *Drosophila*.

A first question which might be asked is: What dose would be required to produce additional mutations equal to those that occur spontaneously? One approach is this: In man those mutation rates that have been measured range from about 4 to 40 per million genes, with an average of about 10 to 15. In mice the average rate of x-ray mutation in several genes studied was 0.25 per million genes per roentgen. Thus, if human genes have the same response to x-rays as those of mice, 40 to 60 r would be required to double the existing mutation rate.

The National Academy of Sciences Committee suggested 30 to 80 r as the range within which the doubling dose is likely to lie, and the British committee chose exactly the same figures. I shall assume 50 r as the doubling dose for further calculations.

The National Academy of Sciences recommended 10 r as a maximum *average* pre-reproductive dose for the population. With a 50 r doubling dose, a population that had been exposed to 10 r every generation for many centuries (long enough to have come to equilibrium) would have an increase of 10/50 or 20 per cent in genetic damage. At present, perhaps 2 per cent of births have some severe genetic defect. A 20 per cent increase would raise this to 2.4 per cent.

Perhaps it is more reasonable to ask the consequences of only one generation of parents exposed to 10 r. On the same assumptions, if there were 100 million births every generation (roughly the present number in the United States), a total of 400,000 new severe genetic defects would be expected. This total would be spread over many generations, with perhaps 40,000 in the first generation. This is about 0.04 per cent of all births.

The writers of the British report attempted figures for specific diseases. They estimated, for example, that doubling the mutation rate would increase by 3 per cent the incidence of low grade mental deficiency in the next generation. Likewise, the first generation increase was estimated as 1 per cent for schizophrenia and 1.4 per cent for manic depressive psychosis. All such estimates, of course, depend on a number of unverifiable, but reasonable assumptions.

As mentioned earlier, it is likely from analogy with *Drosophila* studies that mutations causing severe genetic defects are much less common than those causing small, inconspicuous harmful effects. We can get a crude idea of the cumulative effect of such mutations by considering the total number of mutations of all kinds that might be induced by 10 r. For this, we must rely entirely on mice and *Drosophila*.

In mice the rate of induced mutation is 0.25 per million genes per roentgen. In *Drosophila* the total number of new mutations per fly is about 20,000 times the number at a single gene locus. (This total includes lethals and detrimentals causing as much as a 5 to 10 per cent decrease in viability.) Thus an "organism" with a mouse mutation rate and *Drosophila* gene number would have, if its parents were exposed to 10 r, about five chances in a hundred of having a new mutation.

These mutant genes (5 million among 100 million births) persist in the population until they are eliminated by premature death of some person, or a failure to reproduce. (Some genes, of course, are eliminated by chance, but in a population of constant size these are exactly balanced by those that increase by chance, so that this factor may be ignored.) A fraction, perhaps a large fraction, are eliminated as early embryos, producing relatively little harm. Others may be eliminated only after causing pain or disease to a long descent of persons carrying the gene. Some may contribute to diseases that will be curable by the time the mutant is expressed, so that the harm to the individual may be lessened (though the burden to society may remain great, since the disease persists and must continue to be treated). For all these reasons it is impossible to state what 5 million mutations mean in terms of tangible illness, pain, or decreased life expectancy. But it is quite clear that consideration of only severe genetic defects grossly underestimates the total genetic damage.

COMMENTS

These calculations are quite crude and may be substantially in error. They are based on human data when it is available, but must necessarily depend largely on mice and even on *Drosophila*. One can only guess how comparable these organisms are to man. But unless human genes are grossly more mutable than those of mice (and the Japanese data argue against this), the genetic consequences of 10 r for one generation would be small relative to other causes of death and disease, including spontaneous mutations. For this reason the National Academy of Sciences chose this as a reasonable ("not

harmless, mind you, but reasonable") limit. Nevertheless, our hope is that the average can be kept well below this limit.

Present estimates, necessarily crude, indicate that the average dose received during the first thirty years of life is about 3 r, perhaps more, as a consequence of medical and dental radiation. In the future there will undoubtedly be further increases in the use of radiations and radioactive products, but despite this I hope it will be possible by improved methods to reduce the gonad dose.

It is emphatically not in my province to advise radiologists in the technical aspects of their work. I am not competent to discuss ways of shielding, beam narrowing, and image amplification, or who should and who should not receive diagnostic and therapeutic radiation. As geneticists we are concerned only with the total population dose. We can only ask that all reasonable precautions be taken to keep the dose to the gonads as low as possible. It may well be that we have overestimated the danger, but also we may have underestimated it. Until we know more, it is better to err on the side of over-precaution.

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SUMMARIO IN INTERLINGUA

Consideratione Genetic in le Establimento de Maximos de Dosage de Radiation

Proque doses de radiation non satis grande pro causar apparente insultos somatic pote haber appreciabile consequentias genetic, consideratione genetic deveni factores primari in le establimento de maximos de dosage permissibile pro le population total.

Tres principios genetic es de interesse essential con respecto a iste problema: (1) Mutationes occurre ubique in le corpore e presumibilemente in omne typos de cellulas. (2) Quasi omne le mutationes con effectos satis marcate pro esser detegibile es nocive. (3) Detrimentose genes mutante es eliminate in le curso del tempore ab le population, proque illos reduce le viabilitate o le fertilitate del individuos qui porta los.

Duo principios del mutagenese radiational es etiam pertinente: (1) Radiation augmenta le proportion del mutationes. (2) Le numero del mutationes producite es directemente proportional al dosage de radiation exprimite in roentgens.

Per combinar le tertie principio genetic con le secunde principio radiational, le geneticos conclude que le noxa genetic in le population—non importa como illo se senti o se mesura—es grossiermente proportional al radiation total que es recipite inter conception e reproduction. Isto es ver sin reguardo a como o con qual intensitate le radiation es recipite o a como illo es distribuite inter le membros del population. Si 1 pro cento del population recipe 100 r, le effecto es le mesme como si 100 pro cento habeva recipite 1 r.

In le evalutation quantitative de noxa genetic on debe fider se in grande mesura de studios experimental. Si nos suppone que le observationes experimental es applicabile a humanos, le consequentias genetic de 10 r pro un generation es parve in relation con altere causas de morte e morbo, incluse mutationes spontanee. Isto es le cifra proponite per le Academia National del Scientias como un limite "rationabile."

Considerations Bearing on Permissible Accumulated Radiation Doses for Occupational Exposure[1]

The Aging Process and Cancerogenesis

G. FAILLA, Sc.D.

IN THE PAST it has been assumed that if the weekly radiation dose were low enough, recovery and repair would essentially counteract the tissue damage, and the dose accumulated over a long period of time would become less important, in so far as possible injury manifestable in the lifetime of the exposed individual is concerned (*i.e.*, non-genetic effects). It is becoming increasingly evident, however, that in the case of low-level chronic exposure, there is a not negligible accumulation of effect, related to the total dose over a span of many years. On this account, the concept of limiting the accumulated dose (in addition to limiting the weekly dose) has been introduced into the field of radiation protection. Two of the long-term effects of chief concern are shortening of the life span and cancer production.

Since it would be of considerable help in setting up permissible limits of exposure to have some idea of the processes involved, the writer has given considerable thought to these problems. In the present paper, mechanisms are described, in general outline, to account for the long-term effects mentioned above. These mechanisms involve many assumptions, which, however, are not unreasonable and may well be used as working hypotheses to be substantiated or discarded as more information accumulates. It turns out that the same mechanisms may account at least in part for the spontaneous aging process and carcinogenesis in man. This paper, therefore, is of a more general nature than indicated by the title.

Gross biological effects of x-rays on cells, produced by moderate doses, are influenced by many factors such as oxygen tension, presence of certain compounds (*e.g.*, cysteine), and physiological status of the cell (*e.g.*, time in the mitotic cycle when the radiation is administered, metabolic activity, etc.). Also, in many cases there is some recovery, in the sense that a dose administered over a long period of time is less effective than the same dose given in a short time. On the other hand, when radiation of high specific ionization (*e.g.*, alpha particles, fast neutrons) is used, these modifying factors have little effect on the degree of gross damage produced. This applies also to whole organisms, including mammals.

The normal functions of a cell require synthesis of compounds and removal of waste products, involving many steps. It is clearly demonstrated by the study of biochemical mutants that genes control chemical reactions. Therefore, gene mutations brought about by radiation could interfere with metabolic processes. However, ionization can cause chemical changes more directly. It is reasonable to suppose that with moderate or large doses both modes of action are operative. The difference in the behavior of radiations of low and high specific ionization, may then be explained as follows: Radiation of high specific ionization is most effective in producing chromosome and gene damage; whereas radiation of low specific ionization is most effective in producing diffuse damage throughout the cell. Damage to chromosomes and genes is more drastic and cannot be influenced to any large extent by the modifying factors mentioned above.

In the case of short-term effects, the death of some cells of the organism may well be involved. Long-term effects, how-

[1] Presented at the Forty-second Annual Meeting of the Radiological Society of North America, Chicago, Ill., Dec. 2–7, 1956.

ever, must be due to cells that have been damaged but are still living. They may be considered to be (somatic) biochemical mutants. If these are attributed to gene mutations, it follows that radiation of high specific ionization must be more effective than ordinary x-rays in producing them. It may be concluded further that to produce a gene mutation a group of closely spaced ion pairs is required. This conclusion is contrary to the generally held belief that a single ion pair suffices, but is supported by experimental evidence. The situation is probably as follows: A small number of closely spaced ion pairs is required. In the case of x-rays this occurs at the end of electron tracks (mainly delta rays) or where there is overlap of tracks. Most of the ionization, therefore, is ineffective in producing gene mutations. On the other hand, being diffused throughout the cell, it is capable of producing chemical changes. (Perhaps some of these indirectly produce gene mutations, but this need not be considered at present.) If the dose is given in a short time and is large enough, these chemical changes may cause the cell to die. If the cell does not die, the chemical processes will return to normal (or near normal) because its genetic constitution has not been altered materially. In other words, the cell more or less "recovers."

In the case of radiation of high specific ionization, the ion density along the path of the ionizing particle and at the end of the associated delta rays is sufficient to produce gene mutations. Therefore, a particle passing within a certain distance of a gene produces a mutation. In this sense the process is very efficient but, considering the total energy of the particle, the overall efficiency may still be relatively low. There is, in fact, abundant experimental evidence establishing the low overall efficiency of alpha particles in producing gene mutations. For one thing, this indicates that the ion density along the alpha particle track is probably higher than it need be to produce mutations, *i.e.*, some of the ions are "wasted." It may be surmised that direct interference with the chemical processes going on in the cell is less marked than in the case of x-rays, yet the biological effectiveness of high specific ionization radiation is greater than that of x-rays for short-term gross effects and particularly for long-term ones. It is well known, however, that radiation of high specific ionization is very effective in producing chromosome damage. Hence, this must play an important part in the radiobiological action of this type of radiation. Marked disruption of chromosomes in a dividing cell causes death at the time of division or soon thereafter; in a non-dividing cell it may cause malfunction and premature death. The damage is permanent.

Blair (1), from a careful study of animal experiments (mammals), has pointed out that radiation injury has two components: a reparable one and an irreparable one. In the lethal action of whole-body exposure to x-rays the irreparable component is only a small fraction of the total; in the case of fast neutrons the opposite is true (the reparable component is small). In terms of the mechanism outlined above, the irreparable component is due to somatic gene mutations and probably chromosome damage in all tissues of the body. The reparable component is the result of direct interference with the chemical processes of the cell at one or more links in the chain. In the case of low-level chronic exposure, direct interference with chemical processes may be expected to be practically negligible and therefore only irreparable damage is important. If it is due to gene mutations, it should be proportional to the accumulated dose, which is in accord with Blair's findings.

Effect on Life Span: Shortening of the life span by radiation has been studied by several authors, who agree that in essence it is an acceleration of the aging process. Let us consider the mechanism in terms of gene mutations in the somatic cells of the body. It is assumed that a cell in which one gene has undergone mutation

no longer functions normally. Depending on which gene has been mutated the abnormality may be slight or serious. In a cubic lattice of cells in a tissue, malfunction of one cell is apt to cause some disturbance, however slight, in six cells that are in contact with the first one. It is known that in an organ only a fraction (perhaps 15 or 20 per cent) of the cells are functioning at any one time under ordinary circumstances. In other words, there is a large reserve to take care of occasional increased demands. A considerable fraction of the cells, therefore, could be "incapacitated" without an appreciable effect on the functions of the organ, except in cases of stress. If enough cells are incapacitated, the organ cannot perform the functions required of it to maintain the life of the organism. When the whole body is irradiated more or less uniformly, this applies to all organs, but some, of course, are more vital than others.

Since gene mutations are considered to be rare events, it is necessary to make some estimate of the number of (somatic) cells in which at least one mutation would be produced in each by a certain dose of x-rays. As a starting point we may take the statement in the NAS Genetics Committee report that (roughly) 5×10^6 mutations would be expected in the next 10^8 children if the whole population of the United States now received a gonad x-ray dose of 10 r. One child in 20, therefore, would have an additional mutation. This means that in each parent (at least) 1 germ cell in 40 carried a mutated gene as a result of the 10 r exposure. This would also be true of the somatic cells. Therefore, for a whole body dose of 100 r, at least one cell in 4 would have an additional mutated gene.

Now let us consider spontaneous gene mutation (attributable to background radiation only in part). In the same report it is stated that the doubling dose is probably between 30 and 80 r. For convenience, we may take 50 r in a generation of thirty years. Then in sixty years at least 1 somatic cell in 4 would have a *spontaneous* gene mutation. The presence of so many subnormal cells may well account for the natural aging process. It may be concluded that at conception an individual has a certain inherent life span determined by the genetic constitution inherited from the parents. The actual life span, in the absence of disease, is shorter because of subsequent deleterious mutations. Exposure to ionizing radiation (other than background) accelerates the aging process and further reduces the life span. Disease, of course, may cause acute death. It may also accelerate the aging process by producing additional mutations. This is strongly suggested by statistical data compiled by Hardin Jones (2) and plotted according to the method of Gompertz.

If an individual recovers from a disease, in what way can it have influenced the aging process? There are, of course, many conceivable ways but, for a lasting effect, permanent alterations of cells must have been brought about. These may well be gene mutations caused by mutagenic agents produced by the disease. It is known, however, that an increase in temperature increases the spontaneous mutation rate—a 10°C rise causing a fivefold increase. Therefore, diseases producing fever (*e.g.*, infectious diseases) would be expected to cause additional somatic mutations. If one lets his imagination run wild, there is support for this notion in Hardin Jones's Gompertz curves for Finland and Sweden. It will be recalled that the curve for Finland is at the left of that for Sweden, indicating that at the same chronological age the Finns are physiologically older than the Swedes. One reason for this might be that the Finns are in the habit of taking steam baths (Sauna) which temporarily raise the body temperature! In the same vein, it might be said that the widespread use of aspirin, which reduces fever, has played a considerable part in the prolongation of the life span in the recent past! (More recently antibiotics have, of course, been more effective in this respect.)

Bearing on Radiation Protection Problem: If the aging process is due to the accumulation of mutated cells in all tissues of the body (not necessarily in the same proportions), whatever agents are responsible for the spontaneous mutation rate, then exposure to ionizing radiation simply increases the mutation rate and accelerates the aging process. Hardin Jones (2) has shown, by a wealth of statistical data, that childhood diseases, living standards, and other factors influence the aging process in a systematic way. If the aging mechanism is as postulated above, ionizing radiation acts in essentially the same way. Accordingly, the effect of a certain whole-body dose of radiation on subsequent vitality and life span, may be compared to that of an illness,—say, an infectious disease. This refers particularly to the effect on physiological age as compared to chronological age, in terms of overall death rate. If the mechanism of aging brought about by radiation is essentially the same as that of the natural aging process, we can look to the future with much more confidence in setting up permissible limits of exposure.

Carcinogenesis: The most outstanding characteristic of a cancer cell is its ability to divide indefinitely. In normal tissues in which cell multiplication occurs naturally (*e.g.*, the skin) to take care of wear and tear and to repair damage, cell division is carefully controlled. For instance, if the skin is cut, cell division is greatly accelerated but returns to normal as soon as the damage has been repaired. To account for this, we may postulate that each cell produces an antiproliferating agent which, when present in a certain concentration, prevents cell division. When the skin is cut (or otherwise damaged) the concentration of this agent at the boundaries of the wound decreases and the neighboring cells begin to divide. When healing is complete, the concentration returns to normal. The control of cell division is, therefore, a collective one, *i.e.*, all the cells in a certain volume of tissue contribute to it.

It is not unreasonable to assume that the production of the antiproliferating agent is due to a particular gene in the cell. Therefore, if a suitable mutation of this gene occurs, the agent is no longer produced and the cell acquires the potentiality of indefinite division, *i.e.*, it becomes a cancer cell. It does not follow, however, that a tumor will develop, because surrounding normal cells are able to maintain a sufficient concentration of the agent about the mutated cell. In order for the tumor to develop, a certain number of these normal cells must not function properly or, at any rate, diffusion of the antiproliferating agent from normal cells to the mutated cell must be blocked or interfered with. This may be brought about in a variety of ways (*e.g.*, trauma, aging of cells, etc.), and uncontrolled division starts. Once the number of cancer cells has reached a certain value (*i.e.*, the tumor has reached a certain size) the concentration of the antiproliferating agent at the boundary will always be subnormal and the tumor will continue to grow. It will be seen, therefore, that the establishment of a tumor depends on the accidental juxtaposition of a cancer cell (which has arisen from a mutated normal cell) and a region in which the concentration of antiproliferating agent is, for one reason or another, abnormally low. The juxtaposition need not be simultaneous in time if we assume that the mutated cell can exist for some time. Thus, trauma may cause the development of a tumor in a region in which a cancer cell was already present or, if the trauma persists, a cancer cell may arise there by subsequent mutation of a normal cell.

It has been shown that, if we assume for man the number of spontaneous mutations produced in thirty years to be equal to those resulting from a dose of 50 r, at sixty years 1 cell in 4 would have been mutated at least once. The change to a cancer cell requires (by assumption) a special mutation of a particular gene. If we assume that a human cell contains 25,000 genes and that

the probability of mutation is the same for all (which is not true, but is immaterial to the present argument), 1 cell in 10^5 has been mutated into a cancer cell by age sixty. The number of cells per cubic centimeter of tissue varies with the type of tissue, but for the present purpose we may say that it is of the order of 10^9. It follows that, by the age of sixty, something like 10^4 cancer cells will have been produced per cubic centimeter of tissue. This is a long period of time and, if the life of these *isolated* cancer cells is not long, very few would be present at any particular time. Recalling that the terrain must be propitious before a tumor can develop, the number does not seem to be unduly large. This indeed is fortunate because it indicates that the terrain is the limiting factor and, therefore, an increase in the number of normal cells mutated into cancer cells (*e.g.*, by exposure to radiation) need not bring about a corresponding increase in the incidence of cancer. This is more in line with factual data on the subject. It also permits a more optimistic outlook in cancer control, since it may be assumed that it is easier to modify the terrain than the cancer cell once it has been formed.

It is well to mention at this point that the above number of cancer cells per cubic centimeter of tissue produced in sixty years could be reduced by a factor of the order of 10^4 or 10^5 by assuming that *two particular* genes in the normal cell must be mutated to produce a cancer cell. In sixty years one cancer cell would then be produced in 1 to 10 c.c. of tissue. Further study of the problem is required to distinguish between the two assumptions, if the proposed mechanism is correct in the first place. Many known facts about cancer support the views expressed here and will be discussed in a more detailed paper.

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SUMMARIO IN INTERLINGUA

Considerationes in Re le Permissibile Doses de Radiation Accumulate in Expositiones Occupational

Duo effectos a longe vista que pote esser attribuite al chronic exposition a basse nivellos de radiation es le reduction del duration del vita e le production de cancere. Le mechanismos que es responsabile pro iste effectos es forsan, al minus in parte, identic con illos responsabile pro le invetulation spontanee e le carcinogenese in humanos.

On ha monstrate que noxas per radiation include un componente reparabile e un componente irreparabile, sed solmente le secunde es significative in chronic expositiones a basse nivellos. Illo resulta de somatic mutationes de genes e (probabilemente) de lesiones chromosomal in omne tessutos del corpore. Le accumulation de cellulas mutate in omne tessutos del corpore es etiam postulate como characteristica del processo invetulatori. Le exposition al radiation ionisante augmenta le proportion natural de iste mutationes e accelera le processo invetulatori, i.e. illo reduce le duration del vita.

In le caso de carcinogenese, il es supponite que un gen particular que se occupa de producer un agente antiproliferative es subjicite a mutation con le resultato de libere division cellular e le production de cellulas cancerose. Tamen, le terreno debe esser favorabile pro le disveloppamento del tumor.

Iste observationes es incoragiante in lor signification pro le problema del effectos de

radiation. Si le mechanismo del invetulation que es effectuate per radiation es essentialmente identic con illo del processo invetulatori natural, nos pote reguardar le futuro con plus confidentia quanto al establimento de limites permissibile de exposition. Se presenta etiam un plus optimistic prospecto pro le subjugation de cancere, proque le modification del terreno es un problema plus facile que le modification del cellula cancerose post que illo se ha formate.

DISCUSSION[1]

H. S. Kaplan, M.D. (San Francisco, Calif.): It is probably academic to argue this point but I want to insert a word on the other side of this question. There is really little evidence regarding somatic mutation as a mechanism in cancer induction, though there has been a great deal of talk about it. If the amount of talk were proportional to the evidence there would be a lot of evidence.

Recent evidence points to the existence of indirect mechanisms such as were alluded to by Dr. Dunlap, which are difficult, if not impossible, to reconcile with the somatic mutation idea. It seems to me that we are certainly free to consider the idea of somatic mutation providing we don't let our minds play a trick on us and close the door to all the other possibilities.

Dr. Failla: I agree 100 per cent with the idea that we should not close our minds and work on one hypothesis, that all the possible ways in which cancer might develop should be explored. With the suggestion I have made, you have really quite a lot of leeway in accounting for what is known experimentally in that you have two factors that must be present coincidentally. One is the special mutation that produces the cancer cell, and the other is a propitious terrain for the growth of this cell. Within that range you can explain a lot of things.

Dr. Kaplan: If mutations were really involved here, one might expect the same kind of dose-response relationships that are to be had for gene mutations, namely, irreversibility and additivity, with single doses producing just as much damage as divided, fractionated doses. I think some of the evidence points in the other direction. Most of the skin cancers produced in the martyrs to radiology were the result of small doses, many of which had no effect that was discoverable at any one time. The accumulated doses that some of them got were still rather small, and a single dose of that magnitude would, I think, produce few, if any, cancers. There we have at least a suggestion that multiple, small, divided doses have a disproportionately greater effect in producing cancer. We know from experiments on mice that this is also true for leukemia induction.

Dr. Failla: I don't think it is the proper time and place to discuss this but the two factors I have just mentioned can take care of Dr. Kaplan's example.

Rosalyn Yalow (New York, N. Y.): If one were to accept Dr. Failla's notions concerning the increase in the spontaneous mutation rate, the damage that is produced would not be reversible. It seems to me this would not be easily reconciled with Dr. Jones' statement with respect to the effect of smoking, that ten, twenty, thirty years' effect would be completely reversed merely by discontinuing further injury of that type.

Dr. Failla: That is one of the statements made by Dr. Jones that I don't agree with. I have not looked at his data, but I don't think that conclusion is justified.

Vincent P. Collins, M.D. (Houston, Texas): We have had a most impressive presentation given to us by each of the three speakers, but I am sure the speakers themselves would not like to leave the thought that, although the data stand, the conclusions may not change. The experience of the audience might have something to contribute here. For instance, Dr. Dunlap mentioned the occult findings that might influence the conclusions for a given set of data. There may be a hidden factor governing longevity of radiologists more significant than the aging effect of radiation. Shields Warren reported that the average age at death for radiologists was 60.8 years and for physicians not known to have contact with radiation was 65.7 years. The conclusions that this is related to radiation exposure should be weighed in the light of a tendency that has always existed for medically handicapped physicians to turn to radiology and pathology, which have been believed to offer an easy life rather than a short one.

Milton Friedman, M.D. (New York, N. Y.): I would like to recommend that the maximum permissible dose for radiologists be increased to

[1] This discussion covers not only Dr. Failla's paper, but also those of Dr. Hardin B. Jones (not published), Dr. James F. Crow, and Dr. Charles Dunlap.

five times the present dose, so that they will sustain five times as much leukemia as at present. The reason will be given in a moment.

You all noticed the humility and tentativeness of the statements made by the speakers, which are at variance with official publications contributed to by them and their colleagues and with the government regulations based on information offered by them.

I deplore the absence of a clinical radiobiologist from this discussion because his extrapolations should be as entertaining as the extrapolations of the biologists and statisticians.

Let us examine one of Dr. Hardin Jones' extrapolations, which states that morbidity is the same as mortality. He showed a family of curves drifting to the left, which indicated that average longevity in a population became reduced when the whole body irradiation dose was increased. This is the kind of statement one would expect to find in a booklet on the biologic effect of irradiation of mice, which has been published under the title "Biologic Effects of Atomic Radiation."

The true situation is the following: the larger the amount of irradiation of the whole population up to a certain point, the more the curves should drift in the opposite direction, indicating increased longevity. The reason is that increased irradiation, predominantly from medical diagnostic and therapeutic radiology, reduces disease and consequent morbidity in the population and increases the longevity of man.

This is a specious argument, but it is of the same order of magnitude as the discussions heard today. I offer it with an apology because I know much less about the genetic effects of irradiation on the whole population, the subject of today's discussion, than the speakers. What I am really talking about is the disparity between the amount of true knowledge and the dogmatism of quasi-official and official government regulations concerning radiological protection, which are of increasing importance for the general population. I suggest greater tentativeness in proffering these regulations than exists today.

Dr. Failla: Well, the applause shows where the audience stands on this matter, so perhaps I should not say anything. The thing that bothers me is that many radiologists object to regulations lowering the permissible limits of exposure, and the reason they give is that we don't know exactly what is going to happen, we don't have any good basis for such action. The question I always ask these radiologists is: "what evidence would you want, to be convinced? Or what evidence would be acceptable to you?" Because, if the evidence is based on experiments with flies, the reaction is that there is a big difference between flies and men; if on experiments with mice, that there is a big difference between mice and men. And so it goes, on and on, from which it may be surmised that the evidence they will accept is simply the evidence that some people have been injured or killed by exposure to radiation. Well, people are not being exposed at such high levels, and so you don't see effects, except by statistical studies. There is a statistical study of the survivors of Hiroshima and Nagasaki showing that there is an increase in the mortality rate and in the incidence of leukemia. I think it is accepted even by the radiologists that there is a great increase in the incidence of leukemia in their profession. In Hiroshima and Nagasaki there has been also a greater incidence of cataracts.

Permissible limits, or whatever you want to call them, must be set now, and some estimates or some, let us say, "guesses" must be made as to what might be considered safe. We cannot have the experimental evidence that is acceptable to some radiologists, that is, the human evidence, because by that time it is too late; so we have to make use of whatever evidence is available at this time.

H. M. Parker, M.Sc. (Richland, Wash.): Dr. Taylor planned to make some statements on this morning's fine program but he was unavoidably called away and asked me to substitute for him. I point out that this substitution is definitely non-radiation induced deleterious mutation.

Genetic effects and related layman problems and the question of delayed effects are of vital interest to all of us in this field. I think it is fair to say that some of the speaking and writing in this area either is or appears to be on an emotional or sensational level that reflects discredit on the geneticists or on the radiologists. Dr. Hollaender and those he called on to talk to us this morning are to be congratulated on giving us commonly phrased data in an area in which demonstration that is wholly satisfactory to the scientific instincts of both speakers and listeners is not yet possible.

Genetic Transition as a Determinant of Physiologic and Radiologic Aging and Other Conditions[1]

PAUL S. HENSHAW, Ph.D.

WITH RISING levels of environmental radiation of the ionizing types, and with accumulating evidence that radiation contributes to or accelerates senescence, attention becomes concentrated on aging as an operating process and on the specific role of radiation in this and in other biologic processes. The object of this paper is to take account of *genetic transition* as a means by which both physiologic and radiologic aging may be produced—in part, if not altogether—and at the same time to make certain pertinent correlations.

For the present purposes, aging or senescence is defined as an affliction of the more adult organism, the sequel to which sooner or later is death. Progress of senescence, it is evident, coincides in a general way with passage of time. During the fifth decade of life in man, there begins, as a rule, a noticeable turn toward decreased efficiency of some organs and of the individual as a whole. During the sixth decade, definite reduction in efficiency is apparent almost without exception, and with further passage of time, there comes a cascade of senile changes with death as the end-result—most frequently in about the seventh and eighth decades when individuals live in communities where health standards are maintained at a high level.

Oddly, death is seldom attributed specifically to senility. More often, reference is made to the repressive effects of infection, to the failure of one or another of the organs, or to severe emotional shock, as proving sufficient to strike out or extinguish life in senile persons. Cardiovascular disease, as a terminal condition, correlates with an ever-mounting toll of the aging population, but aging can hardly be defined as progressive deterioration of the cardiovascular system, particularly since it is commonly observed that lower forms without contained circulatory systems also grow old and die.

Analogies can be drawn comparing the process of aging with the wearing out of mechanical devices. A feeble elderly person can be likened to a worn-out automobile motor or to a clock whose parts no longer function properly. That a piston ring should wear out with use and with the passage of time is logical enough and would be expected of a material which is exposed to surface wear and which is unable to reconstitute itself. This picture does not carry over completely to living systems. In some respects the wearing out of a shoe affords a better analogy, inasmuch as wear is inside the parts as well as on the surface, but even here there is lack of parallel.

A fundamental property of living systems—and one, it would seem, that has no counterpart in the world of inanimate objects—is ability to regenerate damaged parts. It is the capacity for reconstitution and autosynthesis, probably more than any other feature, that distinguishes living from non-living things. Recognition of this fact, however, poses a fundamental question or a kind of dilemma, necessitating more detailed consideration of the

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[1] From the Division of Biology and Medicine, U. S. Atomic Energy Commission, Washington, D. C.

Development of this paper occurred during the Summer and Fall of 1956. The paper itself was presented in part at the Argonne National Laboratory meeting Nov. 13, 1956, on "Prediction of Length of Life in Human Populations Following Exposure to Ionizing Radiations," proceedings of which are not being published. The paper by Failla, containing similar ideas, was presented Dec. 5, 1956, at the Forty-second Annual Meeting of the Radiological Society of North America (see p. 23 of this issue of Radiology), but its preparation was started in advance of work undertaken by the present author. It is a pleasure to acknowledge indebtedness to Dr. Failla and Dr. Howard P. Doub for simultaneous publication of the two papers.

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underlying character of life processes. If living organisms can reconstitute themselves, as they do continuously, how can they wear out? With generative tissues, such as mesenchymal elements and germinal epithelium, widely prevalent throughout the body to replenish and rebuild deficient or damaged parts as needed, how is it that an organ or the host organism can grow old?

It is necessary, obviously, to recognize some kind of degradation process, one which involves a progressive lessening of efficiency in most or all of the body parts; also one that has special meaning in relation to fertilization, which constitutes the beginning of life in most multicellular forms, and to atrophy and neoplasia, which so very often are prominent at the end of life. Because of obvious interrelationships, it is natural that steps should have been taken by investigators to search for a unitary principle or a common denominator that underlies these important aspects of life. Features such as nucleic acid metabolism, osmotic fragility, loss of collagen, prevalence of elastic connective-tissue fibers, mineralization of protoplasm, etc., have been investigated, but no really satisfying correlations have thus far been made.[2]

In the paragraphs that follow, an effort is made to examine the case for somatic mutation together with the somewhat overlooked and underevaluated consequent *genetic transition* as the common denominator for a number of biologic manifestations. This will be followed by a series of supplementary correlations, which, it is believed, add strength to the central idea.

PHYSIOLOGIC AGING

Chromosome aberrations and gene mutations, as long understood, appear heterogeneously and with a certain frequency both in the germ line and in the soma, with consequences in the two instances which are comparable in certain respects (2). In the soma, as well as in the germ line, three kinds of mutations occur: the rare and very infrequent *beneficial* type, the deleterious *lethal*, which drop out of the picture very quickly, and the deleterious *sublethal*, which from some points of view are of much greater importance.

Lethal mutations either in the germ line or in the soma lead to early necrosis in some of the cellular descendants. In the germ line this means death of cells affected or abnormalities leading to death in primordia, gametes, zygotes, or embryos; in the soma it means death and loss of a cell here and there throughout the generative tissue components. So far as subsequent situations are concerned, then, such events have little significance other than loss of occasional cells or of some bits of tissue at one stage or another in development. Condition of the species (in case of the germ line) and condition of the organism (in case of the soma) are thus no longer affected by elements that formerly existed.

Sublethal mutations, on the other hand, have a lingering, prolonged and, by comparison at least, much more far-reaching effect, which in the case of the germ line spreads in the population, and in the case of the soma spreads in the organism. As a consequence of sublethal mutations, regions of modified milieu come into being—population islands, so to speak, in the case of regional groupings of people, and tissue islands in the case of somatic cell groupings.

As mutated cells proliferate and spread in the population or in an individual, it is inevitable that the aggregate genetic character should become altered, causing generative elements of the soma as well as of the germ line to consist increasingly of cells with modified genetic potential (3, 4). This transformation is referred to here as *genetic transition*. The term "genetic," as is evident, is used in relation to gene and chromosome dynamics irrespective of whether it pertains to germ line or to soma. From these considerations, it is apparent, and it may be

[2] Some of the thoughts in this and preceding paragraphs stem rather directly from Lansing (1).

accepted, that physiologic *tissue gradients* arise and develop in regions of generative elements of the soma in a manner comparable to the spread of hereditary gradients in a population.

With respect to the germ line, deleterious mutations are eliminated, in time, through natural selection, but in the case of the soma they accumulate as expanding and sometimes diffusing island masses of cells, the number of such masses depending on the frequency of sublethal mutations, and their size depending on time and on the proliferative rate of the cells involved.

Knowing, then, that genetic transformations can and do arise in any tissue type capable of cellular multiplication, and also that the mutations involved may in time be responsible for growing biological weakness in the soma, we recognize the existence of a parallel between that which we take to be inevitable genetic transition, on the one hand, and the aging which we know takes place progressively with time, on the other. This correlation has obvious significance as a basis for explaining physiologic aging, but the parallel goes farther, and various other correlations can be made.

As is well known, mutations may occur spontaneously (*i.e.*, for unknown reasons) and at random in proliferating cellular elements; moreover, they are understood to consist of molecular modifications in nucleoproteins, synthesis of which goes on continuously in multiplying cells. Since beneficial mutations are exceedingly rare, since lethal mutations drop out of the picture quickly, exerting no further influence, and since deleterious sublethal mutations exert an expanding influence, it is evident that a gradual and general degradation takes place in all growing tissues and that termination of life of the soma must be an eventual outcome. In view of these facts, it becomes necessary to look upon aging, at least in part, as the consequence of somatic mutations. Because of the evidence that can thus be brought to bear, question is raised as to whether somatic mutations may not be regarded as responsible.

RADIOLOGIC AGING

Knowing that ionizing radiations induce mutations in proliferating tissues and that most mutations contribute to tissue or organ deterioration, we have, then, a basis for explaining not only how radiologic aging occurs but also how physiologic and radiologic aging can be additive. Mutations have the same biological effects irrespective of the mutagen that produces them, thus making it unnecessary to think of radiologic and physiologic aging as different in so far as the condition of organisms may be concerned.

OTHER AGE-INDUCING AGENTS

Ionizing radiations are but one of several kinds of mutagens, others consisting of heat and biochemical compounds of various kinds. Thus, as a means of developing impressions about somatic mutations as the basis for natural and radiologic aging, it is appropriate and necessary to ask whether life is shortened by other mutagenic agents the same as by radiation.

Evidence is limited, but positive correlations can be made. From experience, it is known that certain infectious diseases cause permanent tissue and organ damage, thereby contributing to an earlier time of death, though the infection has long since been overcome. Here, as in the case of radiologic aging, we are confronted with the question as to how an effect can persist after the noxious agent has been eliminated and repair in the usual sense has become complete—that is, except by means of some permanent alteration of the nucleoprotein materials in cell chromosomes. As in the previous instance, it is obviously necessary to recognize a progressive degradation mechanism, one which in some way involves the cellular biochemical elements that guide the life process.

RADIATION SICKNESS AND LIFE SHORTENING

The concept of somatic mutation and genetic transition as the explanation of aging provides at the same time a logical distinction between irradiation effect lead-

ing to radiation sickness and acute death, on the one hand, and the persisting effect manifested by reduced longevity on the other. It explains also how shortening of life may result from protracted irradiation without being preceded by radiation sickness.

When radiation dosages are large and chromosomal damage extensive (lethal mutation) in all of the self-replenishing tissue elements, the part or organ exposed is in effect sterilized, with the consequence that functional failure and radiation sickness occur. In such a case, if the organs involved are vital, death of the organism also takes place. When dosages are smaller, presumably some loss of cells results from induced lethal changes, but tissue function and tissue replenishment continue through activity of uninjured and slightly injured (sublethally mutated) cells. Organisms that survive acute damage (*i.e.*, the radiation sickness syndrome) have survived the crisis resulting from loss of cells, but they continue to be under the influence of multiplying sublethal mutant cells and as a consequence may die prematurely. According to the idea being developed, radiation sickness would be regarded as primarily the consequence of lethal mutations, since these lead almost immediately to cell loss, and radiation life shortening (the sustained effect after recovery from acute injury) as primarily the consequence of sublethal mutations, inasmuch as such changes comprise a basis for genetic transition and gradual deterioration.

LOCALIZED AGING

A point of importance is the unevenness of aging in different tissues and organs. Radiation, bacterial toxins, chemical agents, and physical injury, any or all of which act to enhance the aging process when applied generally, also can act locally when applied to a particular part. From a large variety of studies with plants and animals, it is known that under these circumstances local mutations are produced. Accordingly it is easy to see why aging or deterioration may be more advanced in one organ than in another; also why death in elderly persons may be due to failure of one organ or organ group in one case and of different organs in another case.

LENGTH OF LIFE AND NATURAL RADIATION

To the extent that aging is explainable on the basis of somatic mutation and subsequent genetic transition, it is necessary to regard mutagenesis as a determinant of length of life and also to regard length of life as governed by rate of mutation and rate of proliferation of mutated cells.

In countries where high standards of public health and safety are maintained and death is more the result of aging than of infection, illness, injury and accident, the average length of life is about seventy years, the Biblical three score and ten. Should it be found that aging is solely the consequence of deleterious mutations, it would follow that, on the average, seventy years is the time required under natural environmental conditions for enough deleterious mutations and enough genetic transition to occur to result in death.

It has been roughly estimated (2) that natural radiation from the earth and cosmic sources accounts for about 16 per cent of the mutations that occur normally in the germ line. The important question is the extent to which natural radiations contribute to "physiologic" aging through induction of mutations. A question closely related, and also awaiting elucidation, is the degree to which variation in mutagenic rate of different species accounts for differences in average length of natural life.

FERTILIZATION AND ITS ROLE WITH RESPECT TO RADIATION EFFECTS

In classic biology, bisexuality and fertilization are emphasized as important in evolution because of the opportunity they afford for diversity of strain types to be achieved through the mixing and spreading of mutant forms in the population.

Fertilization and attendant develop-

ment from a single cell, it may be pointed out, can also perform another function, one little emphasized heretofore. This is a purification or "cleansing" of the cellular elements which maintain the continuity of life. Three kinds of processes are involved: (*a*) starting life anew by the uniting of gametes (or by budding), which allows the soma to be cast off at intervals and by this means eliminating organs which have been transformed to the point of ineffectiveness and inadequacy; (*b*) selecting against badly deformed germ cells (those mutated so extremely that they cannot perform proliferative and other functions effectively) by preventing their maturation to functional ova or sperm; (*c*) selecting against or eliminating zygotes or embryos that arise from gametes which have undergone mutant changes to the point of incompatibility and which lead to death during early cleavage or during prenatal life, thus reducing the number of weakened and deformed organisms that might otherwise be born. Obviously, there are kinds of selection operating in connection with fertilization to maintain or improve the quality of functional cells and thus to influence the efficiency and length of life of organisms. Fertilization, acting in relation to mutation in the germ line and in the soma, it would seem, may be the means by which adherence to tested genetic constitution is accomplished and the achievements of evolution are maintained.

NEOPLASIA

At the other end of somatic life, tumor formation is often a conspicuous terminal condition. Accordingly, as a step in making the picture more complete, it is important in this analysis to consider the possible role of chromosomal changes in carcinogenesis.

Somatic mutation has been put forward for a long time as an attractive explanation of cancer, but in itself has never been found entirely adequate. Standing in the way of its application have been the experimental observations of stepwise changes toward malignancy and of changes that take place progressively and concurrently in large numbers of cells. Each of these findings is in conflict with the idea of a sudden, all-or-none change in a single cell, which so often is visualized as the basis for somatic mutation leading to carcinogenesis. The concept of genetic transition added to somatic mutation in a single cell as the initial step offers a means of rendering theory and observations compatible, an idea pursued to some extent by others (5–7), including Failla in the preceding paper.

From these observations, it is possible to visualize neoplasia as a two-step consequence of somatic mutation. The first step would be mutation, causing change in proliferative function, and the second would be genetic transition, stemming from the single mutation and providing the physiologic field gradient or environmental condition required to elicit expression of malignancy.

The picture developed is consistent with still other features of carcinogenesis. First, there is the fact that radiation-induced tumors arising in adjacent locations in the same germinal tissue may differ widely in type. Beta-ray-induced skin tumors (in rats), though arising from the same germinal epithelium, are rarely alike; each tumor tends to hold to a particular level of epithelial specialization, and these levels differ markedly, covering the entire spectrum of epithelial differentiation (8). This is as would be expected on the basis of random induction of mutations. Second, there is the interval of weeks or months, if not actually years, between events known to cause tumor formation and the time when tumors are detectable. This time requirement correlates with the interval needed for genetic transition, including the malignant manifestations, and it may vary extensively depending upon the nature of mutations induced and the character of physiologic field gradient conditions that exist at different times.

Somatic mutation combined with the

resulting genetic transition, therefore, offers not only a basis for relating aging and carcinogenesis in a consistent and significant way, but also a more consistent characterization of the nature of neoplasia itself.

DUAL CHANNELS FOR RADIATION-INDUCED LIFE SHORTENING

In relation to radiologic life shortening as pictured here, it is important to recognize that this effect is achieved through both the germ line and somatic pathways. Genetic deaths attributable to chromosomal modifications, and occurring earlier than would have been the case if modification had not taken place, result from changes induced in germ cells and spread in the population. Such deaths, depending upon the condition of genetic dominance, may occur immediately in the next generation or later in subsequent generations, but one integrated net effect of all deleterious changes is life shortening. When chromosomal conditions for genetic death are widely prevalent in a population, the average life tenure is necessarily shorter. It is evident, therefore, that mutational changes induced by radiation in the germ line materials of past generations combine with the mutational changes induced by radiation in the soma of the present generation to influence the time of death. Similarly it is evident that radiation applied to the present generation will affect length of life in this generation by action on the soma and in later generations by action on germ line materials.

EVOLUTIONARY PROGRESS

A final point needs to be made with respect to persistence of life in the face of continuous exposure to natural radiation, which is about 0.1 r per year or 3 r per generation. This radiation, it is to be presumed, has produced mutations in both the germ line and the soma; yet it is obvious that evolutionary progress has been made. Knowing then that higher exposure levels reduce vigor and threaten survival, we are faced with the question of threshold levels for deleterious effects.

The following thoughts give some guidance on this question and bring some completeness to the general picture. In case of the germ line, beneficial mutations, though infrequent, are retained and spread in the population, while deleterious, sublethal ones are in time eliminated through natural selection, as we have seen. By retention of the mutations that enable more satisfactory adaptation to the environmental surroundings, and by elimination of those that fail to aid in adaptation, life has been sustained and improvement achieved. It is evident, therefore, that the balance between the rate at which deleterious mutations have been added to the genetic stockpile and the rate at which these have been removed by the selection processes, has been such that evolutionary progress was not prevented.

In the light of this evolving picture, it becomes necessary to consider not only the question of whether background radiation can be increased one to several-fold without harm to genetic quality, but also the question of whether species progress might not be benefited by some increase. While this thought leads away from the idea that all irradiation is injurious to individuals (as opposed to cells), it is not in disagreement with views that the threshold for injurious effects on population groups may be very low—that is, only a few times background. The point of importance is that, while there appears to be no threshold level of irradiation dose for induction of mutations in cells of the germ line, there may, nevertheless, be a threshold for the lowering of genetic quality and species vigor.

SUMMARY

1. Evidence is presented in support of the idea that physiologic and radiologic aging are consequences of somatic mutation followed by genetic transition.

2. Genetic transition is defined as the development of diffusing and overlapping physiologic field gradients of degenerate cells in various tissues and organs as a result of somatic mutation.

3. The thesis is put forward that any mutagenic agent, such as heat, biochemical compounds, and the like, as well as radiations, will contribute to aging.

4. On the basis of randomness and also of localization of mutant changes, an explanation is offered for aging becoming more advanced in one organ than in another, and for differences in organ vitality from one individual to another.

5. Questions are raised: (*a*) as to whether average length of life may not be due in part to the effects of natural radiation; (*b*) as to whether life shortening may not result from effects induced in both the germ line cells and in the soma, and (*c*) as to whether differences in species average life may not be due in part, if not altogether, to differences in mutagenic rate.

6. Fertilization is pictured as a means for cleansing the germ line, and neoplasia as a two-step involvement of somatic mutation.

7. Emphasis is placed on the idea that, while there may be no threshold levels of dose for induction of mutations, there may be such levels for reduction of species vigor.

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SUMMARIO IN INTERLINGUA

Transition Genetic Como Determinante del Invetulation Physiologic e Radiologic e de Altere Conditiones

Es presentate datos in supporto del idea que invetulation physiologic e invetulation radiologic es consequentias de mutation somatic sequite per transition genetic. Transition genetic es definite como le disveloppamento de diffusionante e partialmente coincidente gradientes de campo physiologic de cellulas degenerate in varie tessutos e organos como consequentia de mutation somatic. Es presentate le these que omne agente mutogene—calor, compositos biochimic, etc.—e etiam radiationes avantia le processo invetulatori.

Super le base del distribution al hasardo e etiam de localisation de alterationes mutatori, un explication es offerite pro le phenomeno del plus rapide avantiamento del processo invetulatori in un organo que in un altere e pro differentias del vitalitate de organos inter un individuo e un altere. Es sublevate le questiones (1) si le extension medie del vita non es possibilemente determinate in parte per le effectos del radiation natural, (2) si le accurtation del vita non resulta possibilemente ab effectos inducite tanto in le cellulas germinative como etiam in le soma, e (3) si differentias del duration medie del vita in differente species non es possibilemente le resultato—in parte, si non completemente—de differentias del proratas mutogene.

Le fertilisation es visualisate como un medio de purification o de catharsis del elementos cellular que mantene le continuitate del vita.

Le rolo possibile de carcinogenese es etiam considerate brevemente.

Es sublineate le idea que, ben que il ha possibilemente nulle limine de dosage pro le induction de mutationes, il es ben possibile que tal limines existe pro le reduction del vigor del species.

Gonad Dose During Routine Roentgenography[1]

M. S. BILLINGS, M.D., A. NORMAN, Ph.D., and M. A. GREENFIELD, Ph.D.

SOME TWENTY-FIVE million roentgenographic examinations are performed annually in the United States (1). Thus a substantial fraction of the total population is exposed each year to whatever genetic hazards are associated with these procedures. In order to assess these hazards, it is necessary to know the gonad dose received during each type of examination and the distribution of the examinations in the population. This report presents phantom measurements of the gonad dose received during routine roentgenography together with some statistics of the distribution of roentgenographic studies.

EXPERIMENTAL

The phantom used in these experiments consisted of unit-density Masonite blocks measuring 10.6 × 2.5 × 2.5 cm. Chemical analysis of the material and a computation of the average atomic number, showed it to be tissue equivalent. The phantom was adjusted in all of its dimensions to the average trunk size of adults and children of different age groups (2).

For the measurements, Victoreen thimble chambers of 0.1 r, 1.0 r, and 25 r capacity were used, all of which had been calibrated by the National Bureau of Standards. Removal of a single block from the phantom permitted placement of the dosimeters in appropriate positions. Localization of the female gonads was accomplished with the aid of hysterosalpingograms and pelvic films, in the mid-frontal pelvic plane 9.0 cm. below the surface, 8.5 cm. above the inferior margin of the pubic symphysis, and 4 5 cm. from the mid-line on each side. The male gonad measurements were taken at the caudal mid-point of the trunk phantom 2.5 cm. beneath the anterior surface.

The irradiation technics were those employed in diagnostic radiology at the UCLA Medical Center. Both conventional and high-voltage technics were used. The x-ray factors used for all the measurements were as follows: Target-to-film distance (except for chest) was always 40 inches, this choice being set by the use of a 16-to-1 Bucky grid; for the chest, the distance was 72 inches. Close coning was used in all cases. The added filtration was 3 mm. Al. The half-value layers of the various kilovoltages employed are listed in Table VI.

A small part of the data was derived from measurements on male students submitting to routine chest photofluorography in the UCLA Student Health Department. Calibrated Keleket pocket dosimeters were placed in the gonad region of the subjects anteriorly and posteriorly.

Statistics on the age distribution of roentgenographic examinations were obtained from records of a large private general hospital, a county general hospital, and a children's clinic, all in the Los Angeles area.

RESULTS

Table I gives the gonad dose, central beam dose, and x-ray factors for a number of examinations in children. Because of the large changes in body size that occur in childhood, the measurements were divided into three age groups: 0–2 years, 2–7 years, and 7–11 years. The small body size of children results in gonad doses which are of the same order of magnitude as the central beam skin dose in examinations of the abdomen, spine, and hips. The testes in particular, because of their position, receive a large fraction of the central beam dose.

[1] From the Department of Radiology, School of Medicine and Atomic Energy Project, University of California at Los Angeles. This paper is based on work performed under contract No. AT-04-1-GEN-12 between the Atomic Energy Commission and the University of California at Los Angeles.
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TABLE I: AMOUNT OF RADIATION PER EXPOSURE RECEIVED DURING ROUTINE ROENTGENOGRAPHIC PROCEDURES IN CHILDREN*

	Skull Basal View	Chest AP	Abdomen AP-KUB	Lumbar Spine AP	Lumbar Spine Lateral	Pelvis AP
Infants 0–2 yr.	65 kv., 25 mas.	45 kv., 10 mas.	60 kv., 20 mas.		66 kv., 80 mas.	60 kv., 20 mas.
S	0.18 r	0.023 r	0.16 r		0.8 r	0.16 r
F	0.001 r	0			0.3 r	0.09 r
M	0.001 r	0.002 r	0.15 r		0.8 r	0.16 r
Children 2–7 yr.			50 kv., 50 mas.	60 kv., 50 mas.	70 kv., 120 mas.	46 kv., 50 mas.
S			0.31 r	0.42 r	1.3 r	0.28 r
F	0	0	0.13 r	0.19 r	0.5 r	0.14 r
M	0	0	0.31 r	0.42 r	1.2 r	0.28 r
Children 7–11 yr.			58 kv., 75 mas.		74 kv., 160 mas.	76 kv., 50 mas.
S			0.6 r		1.8 r	0.73 r
F	0	0	0.24 r		0.73 r	0.3 r
M	0	0	0.25 r		0.3 r	0.7 r

Average gonad dose per examination: males, 0.49 r; females 0.29 r.

* The letters S, F, and M refer respectively to measurements on the skin, female gonad region, and male gonad region. Measurements in roentgens were taken with a Victoreen thimble chamber.

TABLE II: DISTRIBUTION OF ROENTGEN EXAMINATIONS IN A CHILDREN'S CLINIC

Number of patients	125
Number of exposures	377
Distribution of exposures	
Sex: Male	54%
Female	46%
Ages: 0–2 years	23%
2–7 years	46%
7 and over	31%
Frequency of Exposures	
Skull	26%
Chest	27%
Abdominal region	15%
Pelvis and hips	22%
Lower extremities	5.5%
Upper extremities	4.5%

In order to calculate the average gonad dose per examination in children, the statistics in Table II were used. These figures on the distribution of exposures according to age and examination were obtained from records of a children's clinic. They indicate an average of three exposures per examination with the distribution shown. Using these data and the gonad doses given in Table I, an average gonad dose per examination in children of 0.49 r in boys and 0.29 r in girls was computed.

Table III gives the gonad dose, central beam skin dose, and x-ray factors for a number of examinations in adults. Measurements were made with both a conventional and a high-voltage technic. As can be seen, the high-voltage technic in general gives a lower gonad dose than the conventional factors.

To compute the average gonad dose per examination in the adult, Donaldson's data (1) were used. Exposures per examination were taken as five in the abdominal region, two in the spinal region, and one in the chest and other parts. This leads to an average of 2.5 exposures per examination, in agreement with the figure given by Donaldson. In this way, the average gonad dose per examination was computed as 0.02 r in men and 0.23 r in women.

For determination of the average dose received by age thirty, which is roughly the mid-point of the reproductive years, use was made of the age distributions shown in Table IV. In a large private general hospital, as can be seen, only 3 per cent of the examined population was less than eleven years old, and an additional 9 per cent was between twelve and thirty years of age. These two age groups represented 22 per cent and 30 per cent, respectively, of the general population (1950 census). Thus, while one-sixth

TABLE III: AMOUNT OF RADIATION RECEIVED PER EXPOSURE DURING ROUTINE ROENTGENOGRAPHIC PROCEDURES IN ADULTS*

	Chest† PA	Abdomen AP	Stomach PA Oblique	Lumbar Spine AP	Lumbar Spine Lat., Rt. Side Up
Low-voltage Technic	62 kv., 13 mas.	62 kv., 100 mas.		72 kv., 100 mas.	78 kv., 200 mas.
S	0.015 r	0.46 r		0.66 r	1.8 r
F	0	0.155 r		0.225 r	0.08 r (l.) 0.48 r (rt.)
M	0	0		0	0
High-voltage technic	120 kv., 0.7 mas.	120 kv., 10 mas.	120 kv., 20 mas.	120 kv., 13 mas.	120 kv., 70 mas.
S	0.003 r	0.21 r	0.36 r	0.25 r	1.5 r
F	0	0.075 r	0 (l.) 0.05 r (rt.)	0.095 r	0.1 r (l.) 0.5 r (rt.)
M	0	0	0	0	0

	Pelvis AP	Salpingogram	Sacroiliac Area AP	Lumbosacral Junction Rt. Lateral
Low-voltage technic	66 kv., 100 mas.	62 kv., 100 mas.		
S	0.5 r	0.5 r		
F	0.2 r	0.2 r		
M	0.5 r			
High-voltage technic	120 kv., 16 mas.		120 kv., 20 mas.	120 kv., 100 mas.
S	0.35 r		0.36 r	2.05 r
F	0.155 r		0.155 r	0.6 r (rt.) 0 (l.)
M	0.35 r		0.05 r	0

Average gonad dose per examination, based on low-voltage technic: males, 0.02 r; females, 0.23 r.

* The letters S, F, and M refer respectively to measurements on the skin, female gonad region, and male gonad region. Measurements in roentgens were taken with a Victoreen thimble chamber.

† T.F.D. was 6 feet for chest films; 40 inches for all other types of examinations.

(25,000,000/150,000,000) of the population receives examinations each year, only $(3/22) \times (1/6)$ in the age group 0–11 and $(9/30) \times (1/6)$ in the age group 12–30 are examined. A total of twelve years is spent in the first group and a total of nineteen years is spent in the second group. Thus, the total dose accumulated through age thirty is given, for males, by: $3/22 \times 1/6 \times 12 \times 0.49 + 9/30 \times 1/6 \times 19 \times 0.02 = 0.15$ r. The corresponding dose for females is 0.3 r. These figures compare closely with estimates for the Australian population of 0.16 r for males and 0.3 r for females (3). They are also in fair agreement with estimates for the British population of 0.128 r for males and 0.193 r for females (4). If the age distributions found in the county general hospital are used (Table IV), the dose received is calculated as 0.73 r in males and 1.1 r in females.

By comparison, the dose received due to naturally occurring radiation, which is about 0.1 r per year (5), gives an accumulated dose of 3.1 r through age thirty. An additional 0.9 r is accumulated from K^{40} in the body, to give a total dose of 4.0 r due to background radiation (5). Thus the contribution of roentgenographic examinations to gonad dose in the first thirty years is probably less than 25 per cent and may be as little as 5 per cent of that due to the naturally occurring background radiation.

Finally, in Table V are listed the results obtained in determinations of the gonad dose received during photofluorography. These suggest a contribution of only 1 to 3 mr per exposure from this source. This in turn suggests that the

TABLE IV: AGE DISTRIBUTION OF ROENTGEN EXAMINATIONS

	Private General Hospital	County General Hospital
Number of patients	251	560
Sex: Male	51%	...
Female	49%	...
Age (years): 0 to 11	3%	15%
12 to 30	9%	30%
31 to 40	8%	14%
41 to 50	15%	12%
51 plus	65%	29%
Average number exposures per examination	5.1 (1,272 exposures)	2.3 (4,701 exposures)

TABLE V: PHOTOFLUOROGRAPHIC SURVEY UNIT

Number of males	480
Dosimeter on skin of lower back of patients (average individual exposure)	8.2 mr
Dosimeter on skin in gonad region in front (average individual exposure)	0.88 mr
Estimated gonad dose per exposure during photofluorography	
Males	1.0 mr
Females	3.0 mr

contribution from photofluorography to the average gonad dose is probably negligible.

DISCUSSION

Ardran (6) has shown that the use of heavy filtration markedly reduces the skin dose per film. For that reason 3 mm. of Al is employed in all radiographic procedures at the UCLA Medical Center. The resulting skin dose is five to ten times less than that reported by other investigators (7). It was felt, nevertheless, that a computation of the average gonad dose received by the population could be based on the measurements made under these conditions, since radiological practice is moving steadily in the direction of lower doses through employment of such improved technics.

This study does not include any estimate of the dose received during fluoroscopy. Very preliminary measurements suggest that fluoroscopy contributes no more to the total gonad dose than does radiography. The same conclusion has been reached by others (7).

The statistical data on the age distribution of examinations were obtained at two hospitals and a children's clinic in Los Angeles. It was hoped that these data would be representative of the distributions to be found in hospitals throughout the United States. Data accumulated by others seem to justify this hope (7). Nevertheless, not only are the distributions quite variable from hospital to hospital, as can be seen in Table IV, but they may change with time as well. The data collected by Moeller *et al.* (8), showing that the sale of x-ray film more than doubled in the decade from 1939 to 1949, is an index to the way the statistics of the distribution of roentgenographic examinations may be changing. Even allowing for the large uncertainty in the statistics used, however, it does not seem likely that, at this time, the contribution of routine roentgenography to the gonad dose received through the age of thirty is more than 25 per cent of that accumulated from the natural background radiations. But these dose estimates would have to be revised sharply upward if prenatal and/or pediatric examinations increase in frequency.

TABLE VI: HALF-VALUE LAYERS AS A FUNCTION OF KILOVOLTAGE

(Initial filtration = 3 mm. Al)

Kvp	h.v.l. (mm. Al)
45	2.2
46	2.2
50	2.4
58	2.6
60	2.7
62	2.8
65	2.9
66	2.9
70	3.1
72	3.1
74	3.2
76	3.3
78	3.3
120	4.7

While the conclusions reached above are generally encouraging, there is, nevertheless, room for improvement. It would appear, in particular, that shielding the gonads of children and young adults whenever possible would decrease substantially the genetic hazards of radiography. This procedure, urged by Stone (9), is in keeping with the sound conservative position that any unnecessary exposure of the gonads is to be condemned (10).

SUMMARY

Measurements of the gonad dose received during routine roentgenography are presented together with some statistics of the distribution of these examinations in the population. It is calculated that the average dose due to these examinations accumulated through age thirty is less than 25 per cent of the dose accumulated

in that time from natural background radiation.

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SUMMARIO IN INTERLINGUA

Doses al Gonades in Roentgenographia Routinari

Pro evalutar le hasardos genetic del roentgeno-examines executate pro objectivos diagnostic, mesurationes del doses recipite per le gonades in varie typos de examine esseva effectuate per medio de modellos e un studio esseva interprendite in re le distribution del examines roentgenographic super le base del protocollos de un grande hospital general private, un hospital general de contato, e un clinica pro juveniles. Le datos assi colligite permitte le calculation que le dose medie accumulate durante le prime tres decennios del vita es minus que 25 pro cento del dose medie accumulate durante ille tempore ab le radiation natural del ambiente.

Le conclusiones del studio es generalmente incoragiante, sed le autores opina que meliorationes additional remane possibile. In particular, il pare que blindar le gonades de juveniles e juvene adultos quandocunque possibile resultarea in un reduction substantial del hasardos genetic del roentgenographia.

Patient Exposure to Ionizing Radiation in Dental Radiography[1]

NORMAN A. BAILY, Ph.D.

THE USE OF ionizing radiations and exposure of the individual are increasing. As larger segments of the population are being exposed, two important requirements become apparent. The first is that exposures to various radiations be accurately evaluated; the second, that all possible means be taken to reduce exposures in necessary medical and dental procedures.

In dental radiography, patient exposure may be reduced by the use of faster films, increased focal skin distance, and more penetrating radiation. The effects of these factors have been considered by various authors. Trout, Kelley and Cathey (1) and Seeman and Cleare (2) have shown that with increased filtration substantial reduction in the dosage to the skin and the first few millimeters of underlying tissue is possible without affecting the diagnostic value of the resulting roentgenograms. The use of such filtration results in a roentgen beam having greater penetration and thereby reduces the ratio of skin to bone dosage. This reduction in skin dose is due to the increase in the ratio of radiation incident upon the skin to that which is useful in producing the x-ray image. The quality of the beam may be further improved by raising the operating potential from the usual 60 or 65 kvp to the range between 85 and 100 kvp.

Increase of the focal skin distance from 8 to 16 inches will further favorably increase the ratio of radiation incident on the skin to that which is useful in producing a radiographic image. For example, if the film is so placed that the beam has traversed 4 cm. before striking it, the increase in radiation reaching it will be 19 per cent.

The employment of ultra-speed film in those cases which will allow its use can further reduce exposure of all tissues irradiated by a factor of four (3).

The object of the investigation to be reported here was to determine the dose delivered to the apical areas during dental radiography, under certain operating conditions. The conditions chosen were the usual operating voltages, i.e., 60 and 65 kvp, added filtration of 0 and 1.0 mm. aluminum, and an 8-inch focal skin distance. The inherent filtration of the machine which was used is 1.2 mm. aluminum (including tube wall, oil, and cone).

METHOD

In order to simulate actual anatomical and geometrical conditions as closely as possible, a phantom head was constructed of Lucite, in sections projected from those given in Eycleshymer and Schoemaker (4). This was done so that all air cavities in the head and neck would be preserved in their relationship to each other and to solid tissue. This phantom, in a typical experimental set-up, is shown in Figure 1.

To accommodate the ionization chamber, six holes were drilled in one side of the head and two holes in the neck. These holes were so cut and shaped that, when the ionization chamber was inserted, essentially no air cavity was present. Three holes were so placed that the center of the sensitive volume of the ionization chamber would coincide with the molar, cuspid, and incisor regions of the maxilla, while three coincided with these same regions in the mandible. The two holes in the neck were made so that the sensitive volume was coincident with each of the thyroid lobes. Removable Lucite pins occupied each hole not being utilized for measurement.

To minimize the variability of beam direction, dots were placed at points corresponding to those anatomical locations at which the cone tip is placed when radiographs are to be taken. These points are

[1] From the Department of Radiation Therapy, Roswell Park Memorial Institute, Buffalo, N. Y. Accepted for publication in January 1957.

illustrated in Figure 18 of *X-rays in Dentistry* (3).

The tube head angulation was that recommended in this same handbook. Although the beam was always applied at the same point, a certain amount of variation in the data recorded could be traced to small shifts in beam direction. As is the general practice, the beam was lined up by eye so that it appeared to be entering perpendicular to a line tangent to the skin at the predetermined field center.

The ionization chamber was placed in the appropriate position and each of the areas was exposed in turn. Dosage rates were recorded for an operating potential of 60 kvp with no added filter and with 1.0 mm. aluminum added filtration. The experimentally determined half-value layers for the two qualities of radiation were 1.0 mm. aluminum and 1.7 mm. aluminum, respectively. The effect of increasing the tube potential to 65 kvp was also investigated. The quality of radiation generated at this potential was 1.2 mm. and 1.7 mm. aluminum, unfiltered and filtered, respectively.

The ionization chamber and associated electrometer were calibrated against a Victoreen "r" meter and chamber which had been calibrated by the National Bureau of Standards. Corrections for wall absorption were those recommended by Day (5).

Field size at the entrance portal was measured photographically. The field was 6.4 cm. in diameter at 8 inches from the target.

Fig. 1. Typical experimental set-up showing phantom head, Lucite block, ionization chamber, and dental x-ray unit as used to determine apical dosage.

RESULTS

Table I gives the total dose administered in the course of a full-mouth examination to the following sites: left and right thyroid lobes; anterior, center, and posterior maxillary areas; anterior, center, and posterior mandibular areas. These dosages were computed on the basis of exposure times listed in Table II (3). The additional exposure sometimes required in the maxillary lateral-incisor region for adults would add somewhat to the figures as listed in Table I. However, since this film is not routinely used, it has been omitted from the tabulated values.

The skin doses delivered when the machine is operated under the different conditions are shown in Table III. With both

TABLE I: TOTAL DOSE DELIVERED BY EXPOSURE OF 14 APICAL FILMS USED IN ROUTINE FULL-MOUTH X-RAY EXAMINATION

Region	Total Dose Delivered (roentgens)			
	60 kvp, with no additional filter		60 kvp, with 1.0 mm. Al. additional filter	
	Adult	Child	Adult	Child
Left thyroid lobe	0.86	0.33	1.1	0.50
Right thyroid lobe	0.86	0.33	1.1	0.50
Anterior maxilla	12	4.9	8.5	3.5
Center maxilla	15	5.9	14	5.6
Posterior maxilla	13	5.1	8.1	3.1
Anterior mandible	23	8.9	16	7.3
Center mandible	22	7.9	17	5.6
Posterior mandible	13	4.4	11	3.9

TABLE II: EXPOSURE TIMES USED TO COMPUTE TOTAL DOSAGE TO AREAS LISTED IN TABLE I

Roentgenogram	Exposure time (secs.) Adult	Child
Maxillary Central-Incisor Region	3	2
Maxillary Cuspid Region	2.5	1.5
Maxillary Bicuspid Region	3	...
Maxillary Molar Region	4.5	2
Mandibular Incisor Region	2	1
Mandibular Cuspid Region	2.5	1
Mandibular Bicuspid Region	2.5	...
Mandibular Molar Region	3	1.25

operating voltages the skin dose is reduced by 1 mm. aluminum filtration to approximately 55 per cent of that when no filter is used.

DISCUSSION

It should be noted that 1 mm. aluminum filtration not only reduces the skin dose by 45 per cent but also lessens the dose absorbed by the bone. This reduction in dosage to the bone structure varies according to the particular area being considered. The minimum reduction recorded for adults was in the anterior maxillary region, amounting to approximately 6 or 7 per cent. The maximum recorded reduction was in the posterior maxillary region, 38 per cent. For children the minimum reduction was to the thyroid area, 6 per cent; the maximum to the rear maxilla, 39 per cent.

When a filter is used, increasing the operating potential from 60 to 65 kvp does not affect the quality of the radiation for practical purposes. This means that the percentage of radiation reaching the film per unit amount of radiation incident upon the skin will not differ with the two kilovoltages. In tests made at this institution, comparable films were obtained for both operating potentials with the same exposure factors.

In calculating the actual dose delivered to the skin of a patient, two factors must be considered in conjunction with the values listed in Table III. First, the use of a field of the size specified will usually mean that a number of areas of the skin are exposed twice to the entrance beam. Second, these areas will be subjected to further radiation as they will also be exit portals for beams entering at other anatomical sites. No attempt was made to determine this dose experimentally because of the difficult geometry and the lack of uniform conditions from patient to patient.

TABLE III: SKIN DOSE ADMINISTERED BY DENTAL X-RAY UNIT UNDER VARIOUS OPERATING CONDITIONS

(Field size, 32.2 cm.^{2}. Initial filtration, 1.2 mm. aluminum)

Operating Potential (kvp)	Added Filter (mm. Al)	h.v.l. (mm. Al)	Surface Dose (r/min.)
60	0	1.03	132
60	1.0	1.71	72.7
65	0	1.15	163
65	1.0	1.75	91.9

In view of the high dosage to the thyroid gland, measurements were made to determine whether this might be reduced by the addition of a protective shield over the thyroid area. Lead shielding of 0.5 to 2.0 mm. was found to result in no appreciable reduction.

CONCLUSIONS

1. For a dental x-ray unit operating at 60 kvp, the addition of 1.0 mm. aluminum filtration (*a*) reduces surface dose by 45 per cent and (*b*) reduces dose to bone and other deep-lying tissues by as much as 38 per cent in some areas.

2. Dose to the bony structure per full mouth set of films ranges from 8 to 23 r for adults and from 3 to 9 r for children, depending on operating voltage and added filtration.

NOTE: The cooperation and advice of Dr. Harold Soloman and Dr. Lenore Simpson are gratefully acknowledged.

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SUMMARIO IN INTERLINGUA

Exposition de Patientes a Radiation Ionisanta in Radiographia Dental

Per medio de un modello de Lucite, le doses de radiation esseva determinate que se delivra, in le curso de un roentgenoexamine dental del bucca integre, al areas anterior, central, e posterior del maxilla e del mandibula e a ambe le lobos del glandula thyroide.

Esseva trovate que le dose recipite per le structura ossee in le curso de un serie complete de expositiones varia inter 8 e 23 r in adultos e inter 3 e 9 r in juveniles, in dependentia del voltage del apparatura e del filtration addite.

In le caso de un apparatura laborante a 60 kvp, le addition de un filtration de 1,0 mm de aluminium al filtration inherente (1,2 mm de aluminium) reduceva le dose al superficie per 45 pro cento e le dose al ossos e a altere tessutos profunde in certe areas per usque a 38 pro cento. Blindar le area thyroide con plumbo non reduceva le dose recipite per le glandula.

Angiocardiographic Aspects of Constrictive Pericarditis[1]

MELVIN M. FIGLEY, M.D., and MALCOLM A. BAGSHAW, M.D.

CONSTRICTIVE pericarditis can ordinarily be recognized on the basis of history, physical findings, and simple laboratory observations (1). Necessary supporting evidence is generally provided by simple roentgen examination (2). The primary physiologic abnormality of reduced stroke volume due to limited diastolic filling can be shown by right heart catheterization (3). Other diseases, chronic heart failure (4), myocardial fibrosis (3), and amyloidosis (5), occasionally present clinical and hemodynamic similarities. Characteristic electrokymographic alterations are described, although these, too, may not be specific (6).

The possibility of direct demonstration of pericardial abnormality by venous angiocardiography provides another approach which has been infrequently applied. Dotter and Steinberg have called attention to the dilatation of the superior vena cava and the thickening of extraluminal soft tissues of the right heart border (7). They stated that the normal soft-tissue band does not exceed 3 mm. in width, but failed to provide the data on which this figure was based. McKusick described these and other less consistent features in 4 cases but considered that they made no contribution to the diagnosis (4). Inasmuch as helpful information has been obtained by angiocardiography in several clinically atypical cases at the University of Michigan Hospital, this review of experiences with the method is submitted.

MATERIAL AND METHODS

The angiocardiograms of 8 patients with surgically verified constrictive pericarditis, 30 individuals with no clinical evidence of pericardial disease, and 6 with pericardial effusion (3 verified by pericardial tap) were studied. The films had been made in postero-anterior projection with the subject sitting at a focus-film distance of 36 inches. Manual injection of the contrast agent (Urokon 70 per cent), serial filming at 1.3 or 2 films per second, and registration of exposure on simultaneous electrocardiogram were used.

The thickness of the right heart wall was observed in each patient. It was generally clearly visualized as a soft-tissue density interposed between the lucency of the lung and the contrast material in the right atrium. Its width was measured at several points (Fig. 1). The point at which the identity of the lateral border of the superior vena cava was lost within the right atrium was labeled *A*. A point just above the cardiophrenic angle was labeled *C*, and point *B* was assigned to the narrowest area between *A* and *C*. Measurements were made on at least three films of each patient, frequently on more.

For estimation of the speed of the intrathoracic circulation, the time in seconds between the appearance of the contrast medium in the superior vena cava and the ascending aorta was noted. The normal range, as determined by this method, has been described by one of us (8).

NORMAL

The extraluminal soft-tissue thickness at point *A* varied unpredictably in the same individual and from one individual to another, but the thickness at points *B* and *C* was approximately equal in the same individual, with only slight variations between exposures. An average value of *B* and *C* was obtained, and a frequency distribution curve plotted. The

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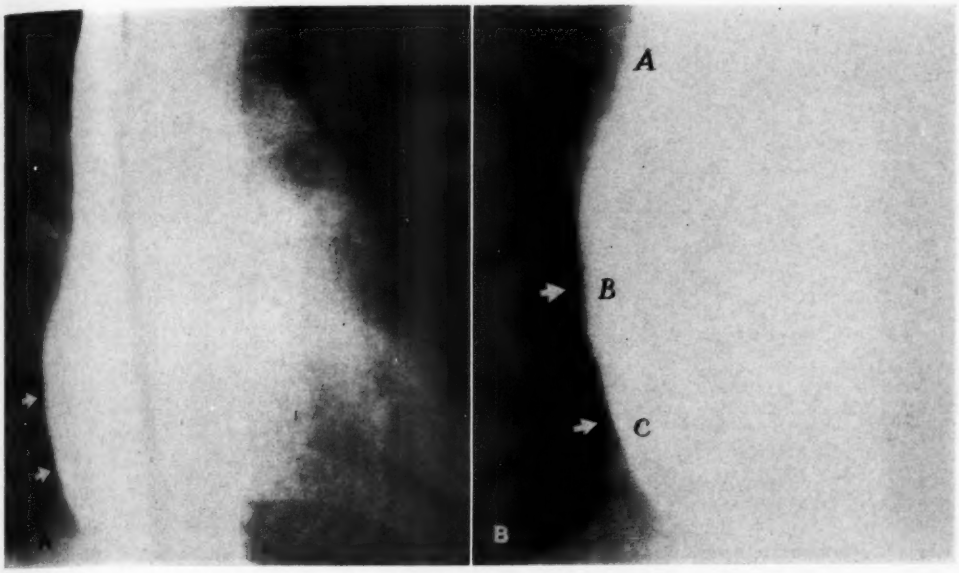

Fig. 1. Normal. A. Extraluminal soft-tissue band (arrows).
B. Enlargement of area of interest showing measurement points, *A*, *B*, *C*.

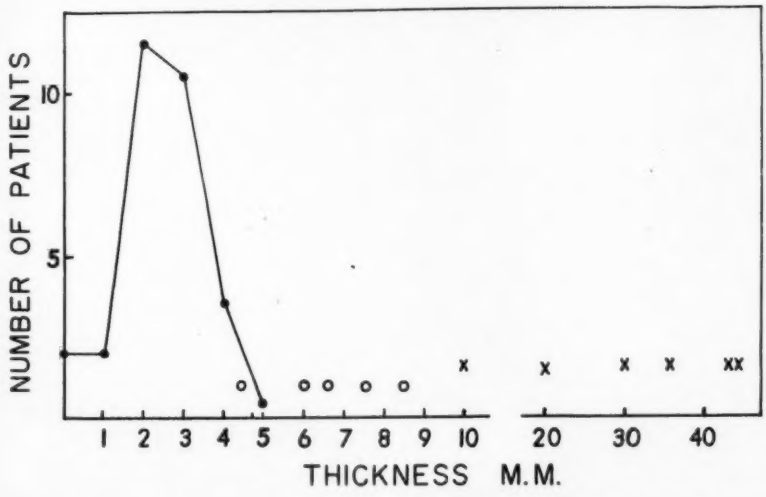

Fig. 2. Extraluminal soft-tissue band thickness. The continuous line is the frequency curve of the normal. The circles (O) indicate observations in constrictive pericarditis; crosses (×) in pericardial effusion.

normal soft-tissue thickness determined in this way does not exceed 4 mm. (Fig. 2).

To be compared later to constrictive pericarditis are certain details of the right border of the right atrial lumen. Normally this was found to vary in position and shape as the result of atrial systole, but it was always convex laterally and parallel to the curve of the overlying heart shadow.

CONSTRICTIVE PERICARDITIS

In 4 of the 5 patients with constrictive pericarditis in whom measurement could

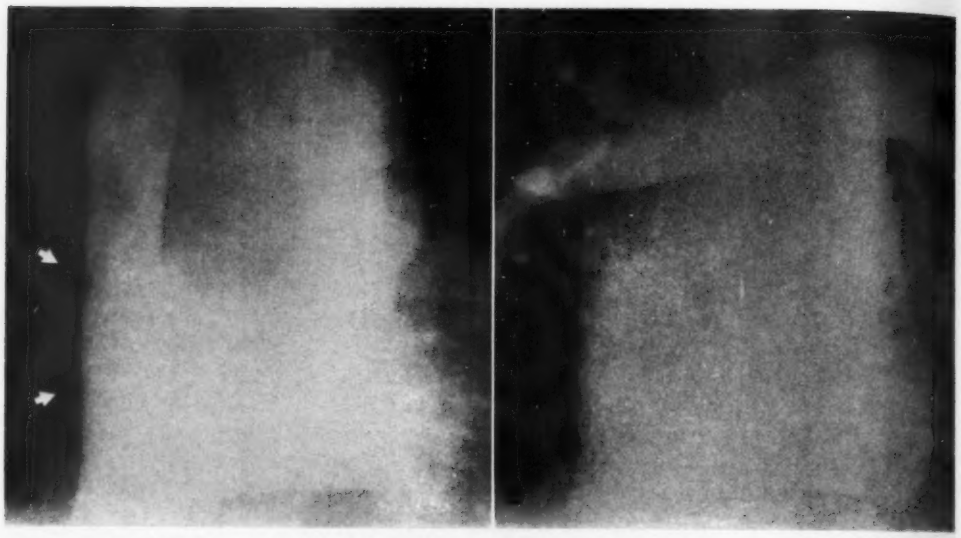

Fig. 3. Constrictive pericarditis. A. The right atrial lumen and superior vena cava have a straight lateral margin. Overlying soft tissues still have a convex contour (arrows). The superior vena cava is dilated.
B. Later, atrial margin unchanged in contour.

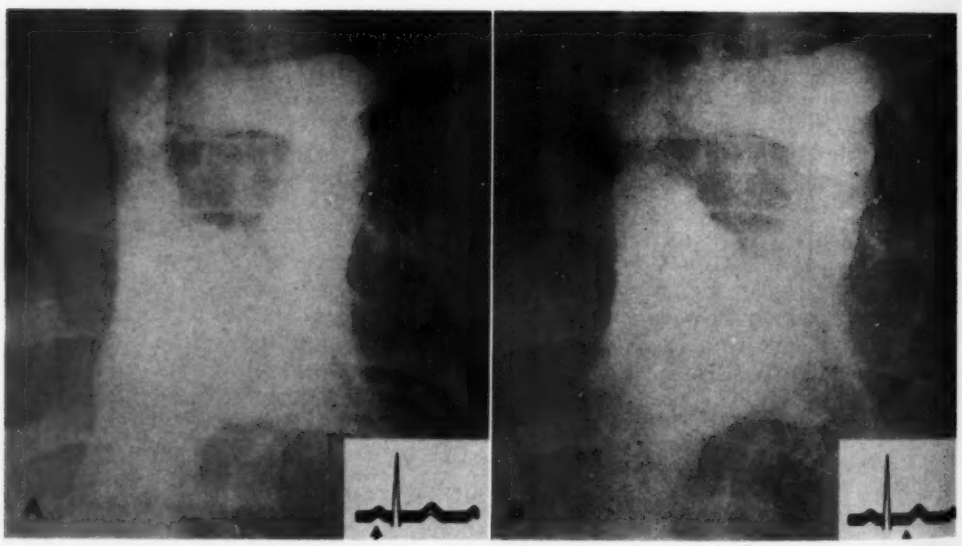

Fig. 4. Constrictive pericarditis. A. Early atrial systole, late ventricular diastole. Note the concavity of the right atrial contour, inferior vena caval dilatation, and small right ventricle. Pleural effusion obscures the soft-tissue-band.
B. Early atrial diastole, mid-ventricular systole. Right atrial margin unchanged.

be made, the extraluminal soft-tissue thickness was clearly increased, varying from 6 to 8.5 mm. In the other it was upper normal at 4.5 mm. (Fig. 2). The values are in line with White's description of thickening of the pericardium of 1 or 2 mm. up to 5 mm. or more (9). In 3 cases measurements could not be made because of obscuration by adjacent pleural effusion in 2 and excessively dark film in 1.

Although interest lay initially in the width of the soft-tissue band in constrictive pericarditis, this review disclosed other abnormalities so consistently that they require equal attention.

1. In 6 of 8 cases the right lateral margin of the right atrial lumen lacked the normal convexity; it continued downward with the margin of the superior vena cava as a straight line (Fig. 3) or was actually concave (Fig. 4). Despite this deformity, some convexity was frequently preserved in the overlying soft tissue.

2. Another unusual feature of the right atrial margin was its striking lack of variation, minor details being reproduced from film to film in a given case. Such seeming rigidity was noted in each of the 8 cases and suggests fixation by the pericardial scar. Rigidity, however, could be proved in only 2 instances, where films were taken in various parts of the heart cycle (Figs. 3 and 4). In 3 cases, heart rate and filming rate were synchronous so that variations, if they did occur, were not shown. In 3 others the presence of auricular fibrillation was itself a cause for invariable contour, since effective atrial contraction was lacking. A related observation, namely dampening or obliteration of right atrial pulsation, was made by McKusick (6) in his electrokymographic studies.

3. Superior vena caval dilatation has been noted by others (4, 7) and was present in 6 of our 8 cases (Fig. 3, A). In 7 cases reflux of contrast material downward, a feature seldom observed in the normal, demonstrated striking localized dilatation of the inferior vena cava (Fig. 4, A). Vena caval constriction was not present in any case, further supporting the well documented fact that it is not the mechanism of the disease (3, 4, 7).

4. Prolonged circulation time was a feature in all but one patient (Fig. 5).

5. Supporting the thesis that restriction of diastolic filling of the ventricles is the primary mechanism in this disease is our observation that the diastolic size of the right ventricle was not definitely enlarged in any case and seemed smaller than normal in 3 (Fig. 4, A). Admittedly the observations are not well controlled, as no normal standards are yet available. The left ventricle was seldom seen well enough for estimation of its size. An appraisal of stroke volume of the ventricles was seldom possible because of insufficiently rapid filming and lack of normal standards, but in one patient the variations in the cavity size of the left ventricle were surprisingly large (Fig. 6, B and C).

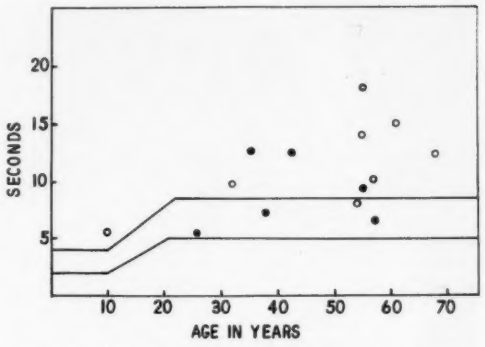

Fig. 5. Angiocardiographic circulation time between superior vena cava and ascending aorta. Continuous lines demarcate the normal range. Open circles (○) indicate observations in constrictive pericarditis; black dots (●) in pericardial effusion.

6. Left atrial enlargement has been described in constrictive pericarditis at necropsy (4) and radiologically on the basis of esophageal displacement. This feature and an occasional murmur may suggest mitral stenosis (4), although the combination of these diseases is uncommon (9). In 3 of our cases there was slight but definite enlargement of the left atrium as judged by its diastolic size (Fig. 6, B). These same cases were the only ones to show any right atrial dilatation. If Sawyer's interpretations are correct, all patients had equally elevated pressures in both atria (3) and on this basis atrial dilatation might be expected regularly. Presumably the scar restricted such dilatation in most cases, even though the left atrium is partly extrapericardial in position. The patient with the largest left atrium was also clinically regarded as having mitral valve disease.

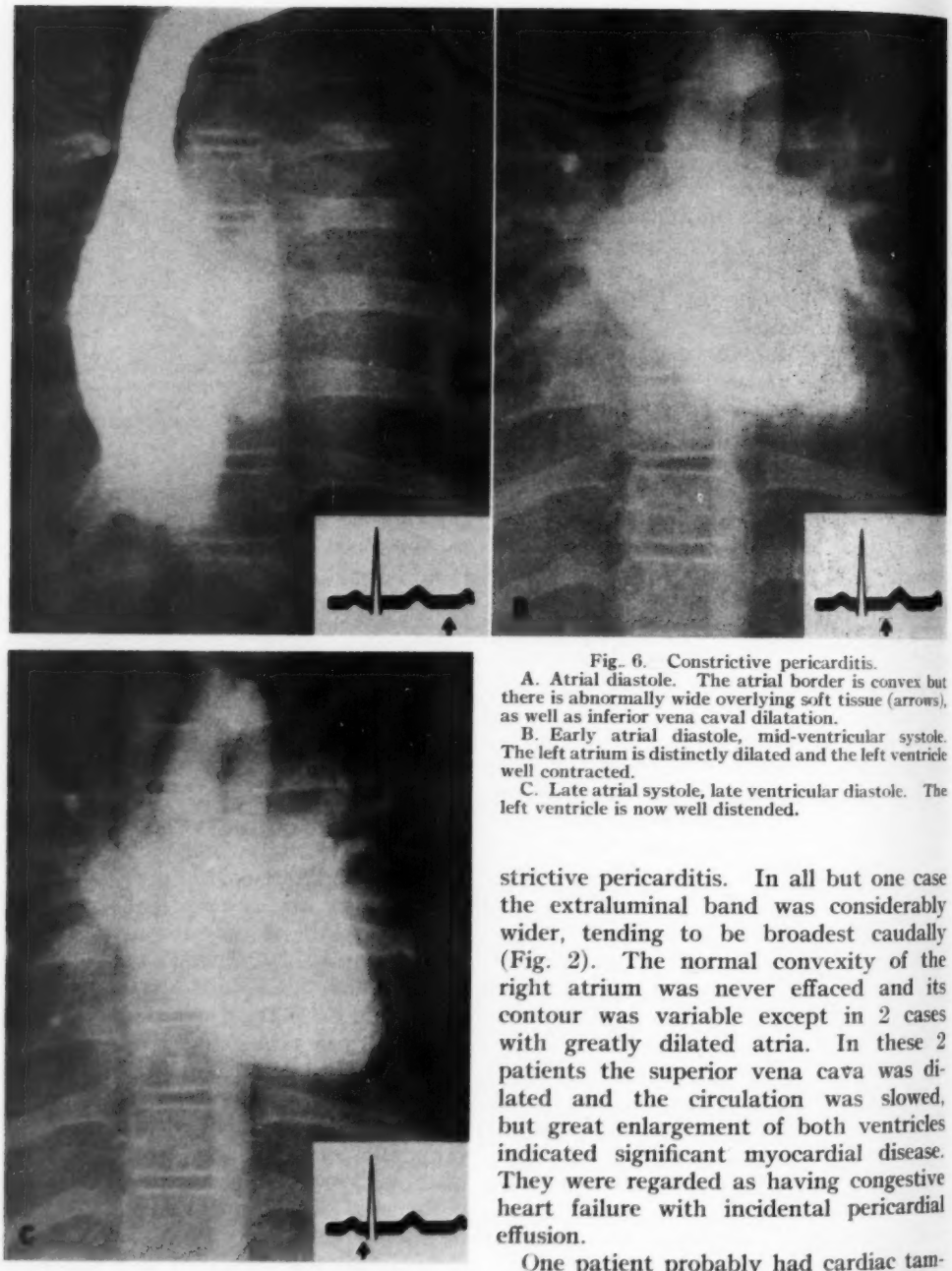

Fig. 6. Constrictive pericarditis.
A. Atrial diastole. The atrial border is convex but there is abnormally wide overlying soft tissue (arrows), as well as inferior vena caval dilatation.
B. Early atrial diastole, mid-ventricular systole. The left atrium is distinctly dilated and the left ventricle well contracted.
C. Late atrial systole, late ventricular diastole. The left ventricle is now well distended.

PERICARDIAL EFFUSION

In comparison, it may be noted that the 6 patients with pericardial effusion varied in significant detail from those with constrictive pericarditis. In all but one case the extraluminal band was considerably wider, tending to be broadest caudally (Fig. 2). The normal convexity of the right atrium was never effaced and its contour was variable except in 2 cases with greatly dilated atria. In these 2 patients the superior vena cava was dilated and the circulation was slowed, but great enlargement of both ventricles indicated significant myocardial disease. They were regarded as having congestive heart failure with incidental pericardial effusion.

One patient probably had cardiac tamponade. He showed no atrial deformity or rigidity or vena caval dilatation, but did have slightly elevated circulation time. In the 3 remaining cases, in which the pericardial fluid seemed to cause no

disability clinically, the circulation time was normal.

DISCUSSION

The soft-tissue layer under discussion is composed of visceral pleura, the parietal pleura, the pericardium, the epicardium and its fat, the right atrial myocardium, and the endocardium. Theoretically, thickening of any of these components or fluid in the pleural and pericardial spaces could produce widening of the soft-tissue band as seen by angiocardiography. Also, a well aerated segment of lung closely applied to the right heart border is necessary for accurate evaluation. It is usually available, although coincident disease in the right middle or lower lobes, or an especially high right diaphragm, could complicate interpretation.

Paramediastinal pleural effusion was the cause of difficulty in measurement in 2 cases. Since it is often present in constrictive pericarditis, it is advantageous to withhold angiocardiography until the fluid has been removed by thoracentesis or diuresis. In the 2 cases mentioned, right atrial deformity was none the less demonstrated, allowing a presumptive radiologic diagnosis (Fig. 4).

A small pericardial effusion may likewise widen the soft-tissue band but, as already indicated, the accessory findings of vena caval dilatation, straightening and fixation of the right atrial border, and delayed circulation are not likely to be caused by a small effusion alone. A pericardial neoplasm has been described (10) as widening this band, but the band was irregular and wider than in the cases of constrictive pericarditis presented here.

Right atrial hypertrophy sufficient to be confusing has not been encountered. A case of cardiac amyloidosis, seen recently at necropsy, showed little if any thickening of the atrial wall. On one occasion, rotation of the heart brought the right ventricle into profile along the right heart border, widening the band, but the trabecular pattern readily allowed its identification. In another case, with no clinical signs of constrictive pericarditis, widening of the extraluminal band was presumably due to unusually thick pericardial fat (Fig. 7). This was notably thicker in the cardiophrenic angle.

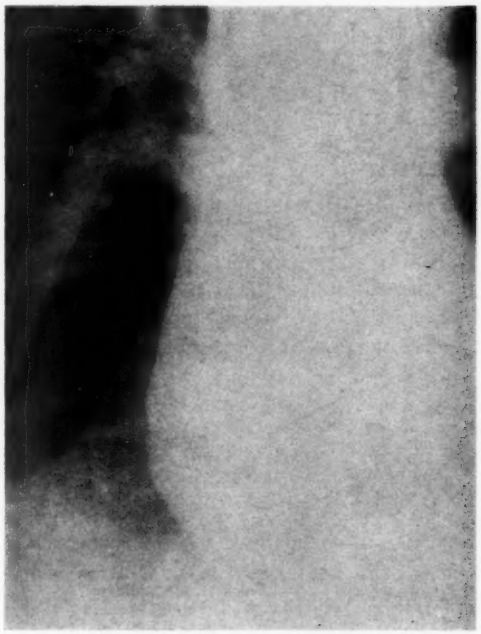

Fig. 7. Pericardial fat. Male, 55, with no symptoms or signs of cardiac disease. Wide extraluminal band considered to be thickened pericardial fat. No proof.

A schematic representation of the right heart border as seen by angiocardiography (Fig. 8) demonstrates the characteristic patterns of the normal, constrictive pericarditis, pericardial effusion, and pericardial fat.

On occasion, large pericardial effusions have been recognized by the abnormally wide separation of the cavity of the left ventricle from the left heart border (11). For the detection of lesser degrees of thickening that occur in constrictive pericarditis, the left side is not so satisfactory as the right because the left ventricle is not always adequately opacified and because its wall thickness varies so greatly throughout the heart cycle (Fig. 6, B and C). Only the late diastolic appearance can be considered if reliable observations are to be obtained.

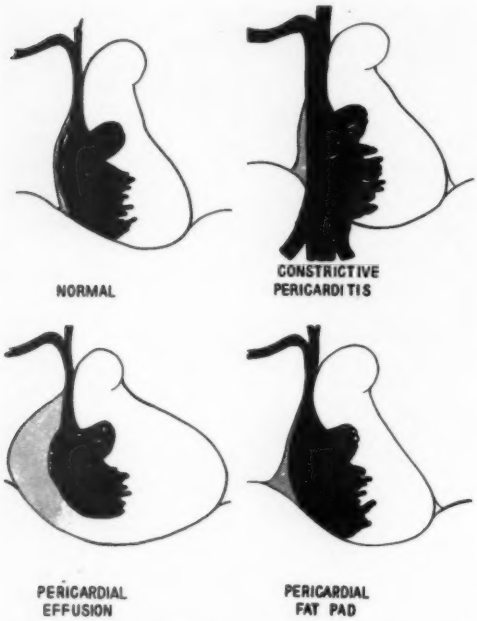

Fig. 8. Schematic diagram of angiocardiographic details of the extraluminal soft-tissue band, vena caval and right atrial contours.

Although certain features help differentiate the various causes of thickening of the right border soft-tissue band, the diagnosis of constrictive pericarditis cannot rely on this sign alone. Emphasis must be placed on the value of the accessory signs, particularly the fixation and deformity of the right atrial border, vena caval dilatation, and slowed circulation in the presence of a normal-sized right ventricle. If these are present to support the observation of a widened extraluminal band, a diagnosis of constrictive pericarditis may be confidently suggested by angiocardiography. By no means does angiocardiography discredit the simple diagnostic methods, but when these fail in the atypical case it may yield graphic and reliable evidence of the disease.

SUMMARY

1. By venous angiocardiography the thickness of tissues of the right heart border was demonstrated in 30 normal patients, in 5 with constrictive pericarditis, and in 6 with pericardial effusion.

2. Normally this border rarely exceeds 4 mm. in width. It varied from 4.5 to 8.5 mm. in constrictive pericarditis and 10 to 15 mm. in pericardial effusion.

3. Accessory angiocardiographic signs of constrictive pericarditis are straightening and rigidity of the right atrial border, superior and inferior vena caval dilatation, slowing of intrathoracic circulation, a right ventricle of normal or small size, and slight left atrial dilatation.

4. These observations in the presence of a consistent clinical picture allow a reliable diagnosis of constrictive pericarditis.

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SUMMARIO IN INTERLINGUA

Aspectos Angiocardiographic de Pericarditis Constrictive

Per medio de angiocardiographia venose, le spissitate del tessutos del margine dextero-cardiac esseva demonstrate in 30 patientes normal, in 5 patientes con pericarditis constrictive, e in 6 patientes con effusion pericardial. Sub conditiones normal, iste margine es rarmente plus que 4 mm in largor. In pericarditis constrictive illo variava inter 4,5 e 8,5 mm e in effusion pericardial inter 10 e 15 mm.

Accessori signos angiocardiographic de pericarditis constrictive es rectitate e rigiditate del margine dextero-atrial, dilatation de vena cave superior e inferior, relentation del circulation intrathoracic, un ventriculo dextere de normal o parve dimensiones, e leve dilatation sinistro-atrial.

Iste observationes in le presentia de clar corroboration clinic permitte un benfundate diagnose de pericarditis constrictive.

Radiologic Aspects of Operable Heart Disease

IV. The Variable Appearance of Constrictive Pericarditis[1]

RANDALL HEINZ, M.D., and HERBERT L. ABRAMS, M.D.

IN PREVIOUS publications, the radiologic aspects of operable congenital anomalies have been stressed (1–3). One relatively rare acquired lesion in which significant surgical relief may be afforded is constrictive pericarditis. Because recognition of this disease is sometimes difficult, a review of proved cases was undertaken, in order to evaluate the usefulness and consistency of the roentgenologic features.

METHODS AND MATERIAL

Twenty-one cases, all proved at surgery and all studied intensively, both clinically and radiologically, were analyzed. The clinical, laboratory, and pathologic data for each case were summarized, and the conventional roentgenograms and kymographic studies were re-examined, with careful attention to previous fluoroscopic findings. Notations were made as to overall heart size, cardiothoracic ratio, individual chamber size, central and peripheral pulmonary artery size, the presence or absence of pulmonary engorgement, the prominence of the superior vena cava and aorta, the presence, extent, and location of pericardial calcification, and the cardiac pulsations as noted at fluoroscopy and in kymograms.

RESULTS

1. *Etiology:* In 5 cases, tuberculosis was undoubtedly the cause of the pericardial disease. A sixth patient gave a history of an acute illness, characterized by chest pain and fever, twelve years earlier. The diagnosis of rheumatic fever was made at the time, but no signs of valvular disease were present on admission and calcified hilar nodes were demonstrable roentgenographically.

In 14 cases there was no history of tuberculosis, chest injury or acute pericarditis. The tuberculin skin test was performed in 9 of this number and was positive in only 1 instance. One patient had associated congenital pulmonic stenosis. In his case, the development of acute pericarditis, subsequent effusion, and finally of a constrictive pericarditis could be clearly traced over a period of about a year (Fig. 1). Two patients had well documented histories of rheumatic fever and had rheumatic valvular disease.

In 1 case, a substernal abscess had been drained ten years prior to the onset of signs and symptoms of constrictive pericarditis. This patient had no evidence of tuberculosis, and a pyogenic pericarditis was presumed to be the cause of the pericardial thickening and constriction.

2. *Pathology:* The specimens of surgically removed pericardial tissue examined microscopically consisted mainly of acellular, dense fibrous tissue with variable degrees of hyalinization. Ten cases showed calcium in the tissue. Except for 5 instances in which acid-fast bacilli and/or tubercle formation were noted, no variations were observed from which a clue to etiology could be obtained. The thickness of the pericardial tissue was between 2 and 4 mm.

3. *Signs and Symptoms:* Before reviewing this portion of the histories, 3 cases of active tuberculosis and 1 of congenital pulmonic stenosis were eliminated for obvious reasons. Seventeen cases remained to be considered.

The early symptoms noted were ascites in 14 instances, dyspnea in 12, ankle edema in 10, and orthopnea in 1. The time from the onset of symptoms until surgery ranged from two months to twelve years, with an average of forty-two months.

[1] From the Departments of Medicine and Radiology, Stanford University School of Medicine, San Francisco, Calif. Accepted for publication in January 1957.

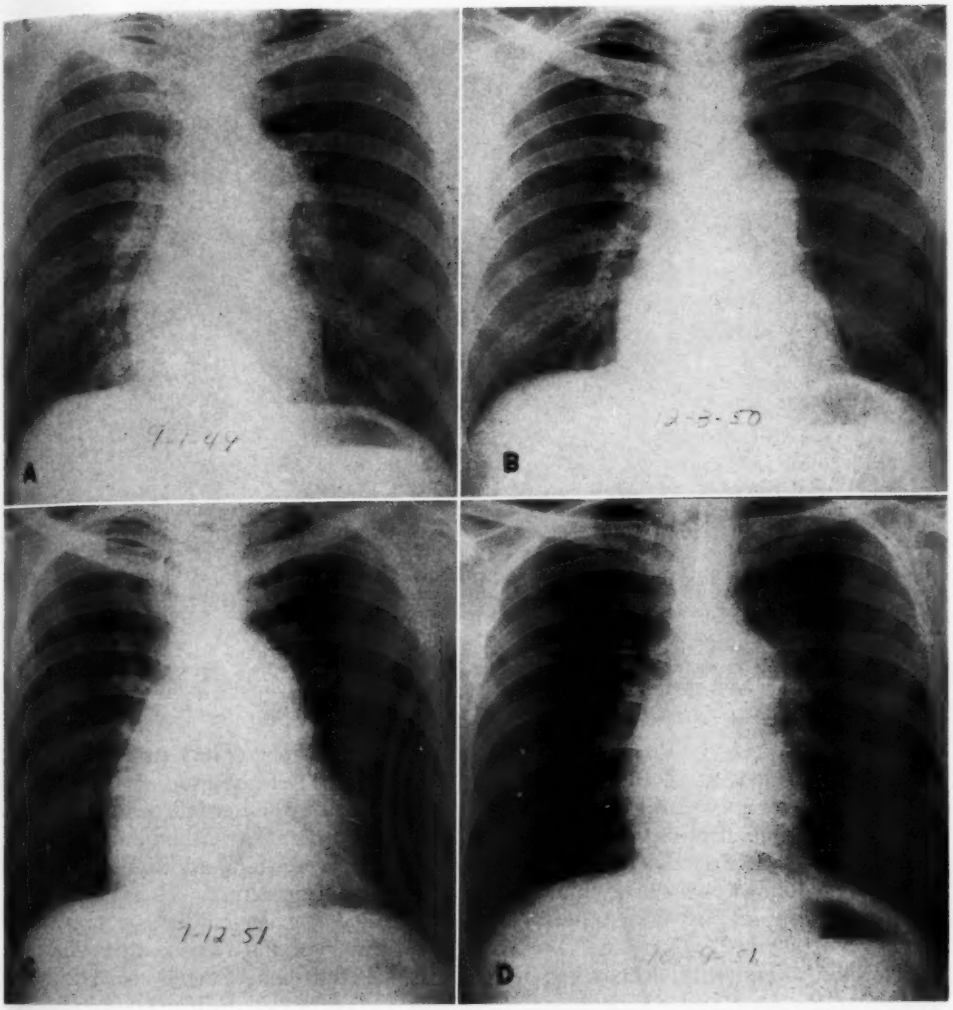

Fig. 1. The development of constrictive pericarditis. This 26-year-old man had noted episodes of palpitation and dyspnea since childhood. At the age of eighteen, he was rejected by the Army because of a "bad heart." Some years later he had a prolonged episode of fever and chest pain accompanied by enlargement of the cardiac silhouette. Thereafter, increasing venous distention and ankle edema developed. Cardiac catheterization demonstrated the presence of pulmonic stenosis. At surgery, marked fibrous thickening of the pericardium was found, and pericardiectomy was performed. In addition pulmonic valve stenosis was noted and a valvotomy was done.

A. 1949. Prominence of the pulmonary artery segment is apparent, due to post-stenotic dilatation.
B. 1950. Some apparent cardiac enlargement has developed.
C. July 1951. Gross enlargement of the cardiac silhouette due to pericardial effusion is present.
D. October 1951. The effusion has resorbed, and the appearance of the heart is not markedly different from that of 1949 (A).

Distention of the neck veins in the sitting position was present in 14 cases; the venous pressure varied from 153 to 420 mm. saline, with an average of 243 mm. saline. The arm-to-tongue circulation time, determined in 15 cases, varied from seventeen to thirty-five seconds, with an average of twenty-four seconds.

A paradoxical pulse was noted in 7 cases, and in 11 cases the heart sounds were diminished. On admission, the liver was enlarged in every instarce.

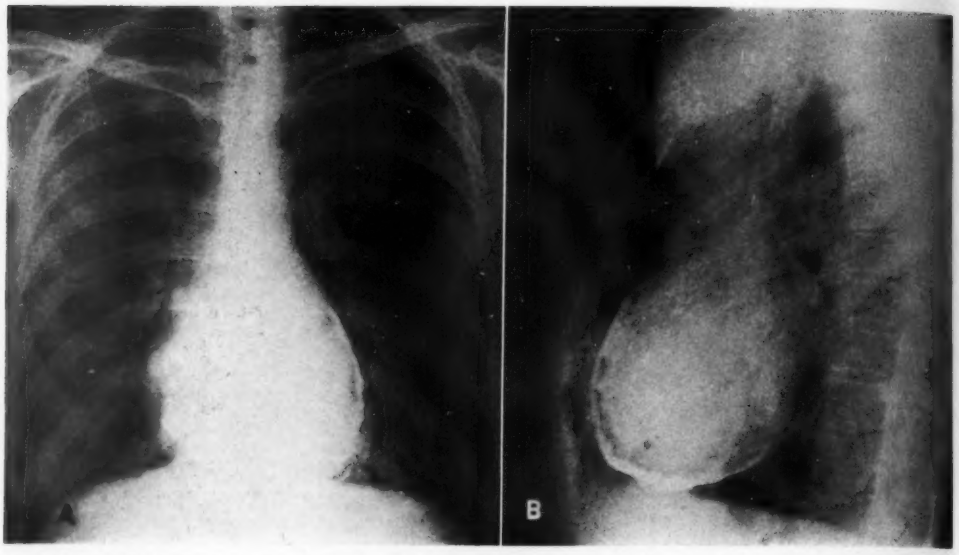

Fig. 2. Calcific constrictive pericarditis with normal cardiac size. This 49-year-old woman developed ankle edema and dyspnea four years prior to admission, with increasing severity of symptoms. Calcification of the pericardium was noted six months before admission. At surgery, the pericardium was grossly thickened and visibly calcified.

A. Postero-anterior projection. Calcification along the left, diaphragmatic, and right heart border is visible. The pulmonary vessels are essentially normal, but there is slight dilatation of the superior vena cava.

B. Left anterior oblique projection. The extent of the calcification is now clearly demonstrated.

4. *Laboratory Data:* Preoperative electrocardiograms were available in 18 cases.[2] Seven of the tracings showed low voltage (the main QRS deflection was 5 mm. or less in the standard leads) and 3 showed prolonged P-R intervals. Auricular fibrillation was present in 4 instances, and P-wave changes in 2. Abnormal T waves were noted frequently, occurring most commonly in the standard leads and the left precordial leads.

Cardiac catheterization studies on 5 patients demonstrated moderately elevated right atrial, right ventricular, and pulmonary artery pressures in all. The cardiac output was low. In none of the 5 were there significant pressure gradients between the venae cavae and the right atrium, the right atrium and the right ventricle, or the right ventricle and the pulmonary artery. A sixth patient was found, on catheterization, to have a right ventricular pressure of 50/13 mm. Hg., and a pulmonary artery pressure of 19/15 mm. Hg. That the gradient was not due to a localized external constriction was shown at operation; pulmonic valve stenosis was present in addition to the constrictive pericarditis.

5. *Roentgenologic Findings:* (*a*) *Overall heart size.* Among 16 patients in whom the size of the heart could be adequately assessed, it was within normal limits in 8 (Fig. 2), slight to moderately enlarged in 4, and grossly enlarged in 4. Half of the patients had a cardiothoracic ratio above 0.50. This gross method of cardiac mensuration was employed because it seemed worthwhile to assess the validity of the usual description of a "small heart" in this condition; obviously a significant number of patients did not appear to have a small heart (Figs. 3 and 4).

(*b*) *Individual chamber size.* In a few cases, chamber size was difficult to evaluate because of bilateral pleural effusions. Right atrial enlargement was thought to be present in 3 of 17 cases, in 1 of which, it

[2] The electrocardiogram of the patient with pulmonic stenosis is not included in this study.

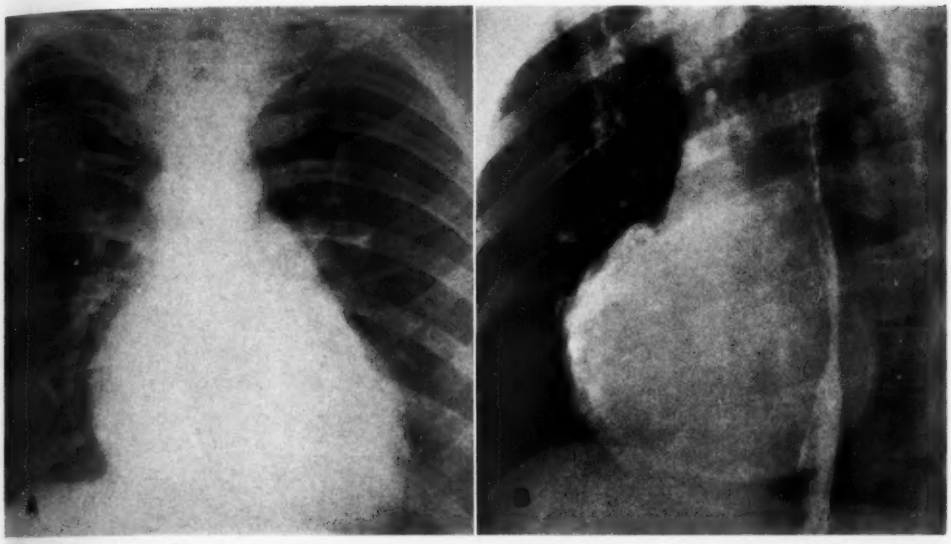

Fig. 3. Cardiac enlargement in calcific constrictive pericarditis. This 33-year-old woman had experienced severe dyspnea during two pregnancies. The dyspnea had increased thereafter and hepatic enlargement was noted. At fluoroscopy, cardiac pulsations were markedly diminished.

A. Postero-anterior projection. Definite cardiac enlargement is present. Calcification along the left heart border is barely visible. There are dilatation of the superior vena cava (arrow) and some pulmonary engorgement.

B. Left anterior oblique projection. The calcific envelope around the right ventricle, left ventricle, and diaphragmatic border is far more extensive than the postero-anterior view would lead one to believe. Definite enlargement of the right cardiac chambers is apparent.

was part of a generalized cardiac enlargement. In another of these cases, pulmonic stenosis was present. Right ventricular enlargement, as evaluated in the left and right anterior oblique projections, was noted in 11 cases (Fig. 3). Left atrial enlargement was apparent in 3 patients, in one of whom there was coexistent rheumatic heart disease with mitral valve involvement. In only 3 patients was there definite left ventricular enlargement.

(*c*) *The pulmonary arteries.* Fourteen cases showed significant prominence of the pulmonary artery segment of the left heart border (Figs. 3, 4, A and C). In 9 of these, the prominence was moderate to marked. The central or hilar pulmonary arteries were increased in prominence in 16 cases, the peripheral pulmonary arteries in 8. Moderate to marked pulmonary engorgement was noted in 11 cases.

(*d*) *The superior vena cava.* In 11 cases, the superior vena cava seemed unduly widened (Figs. 3, A, 4, A, C, and D). In a significant group of these, the azygos vein was also noted as a shadow of increased size adjacent to the point of origin of the right main stem bronchus.

(*e*) *The aortic knob.* The aortic knob seemed normally prominent in 13 cases (Figs. 2 and 3) and hypoplastic in 5.

(*f*) *Pericardial calcification.* In 11 of 19 cases, calcification of the pericardium was demonstrable roentgenologically (Figs. 2, 3, 4, D); in 8 it was gross and extensive. The commonest sites, in descending order, were the anterior or right ventricular surface, the diaphragmatic surface, the left ventricular or posterior surface, the right atrial surface, and the left atrial surface. In 2 cases calcification was limited to the atrioventricular groove (Fig. 4, D). In some instances the calcification was plaque-like and discontinuous; in others it formed an envelope around the heart (Fig. 3, B). Although the heart size tended to be somewhat less in those cases with the most extensive calcification (Fig. 2, A), there was no sharply defined correlation between these two features (Fig. 3, A).

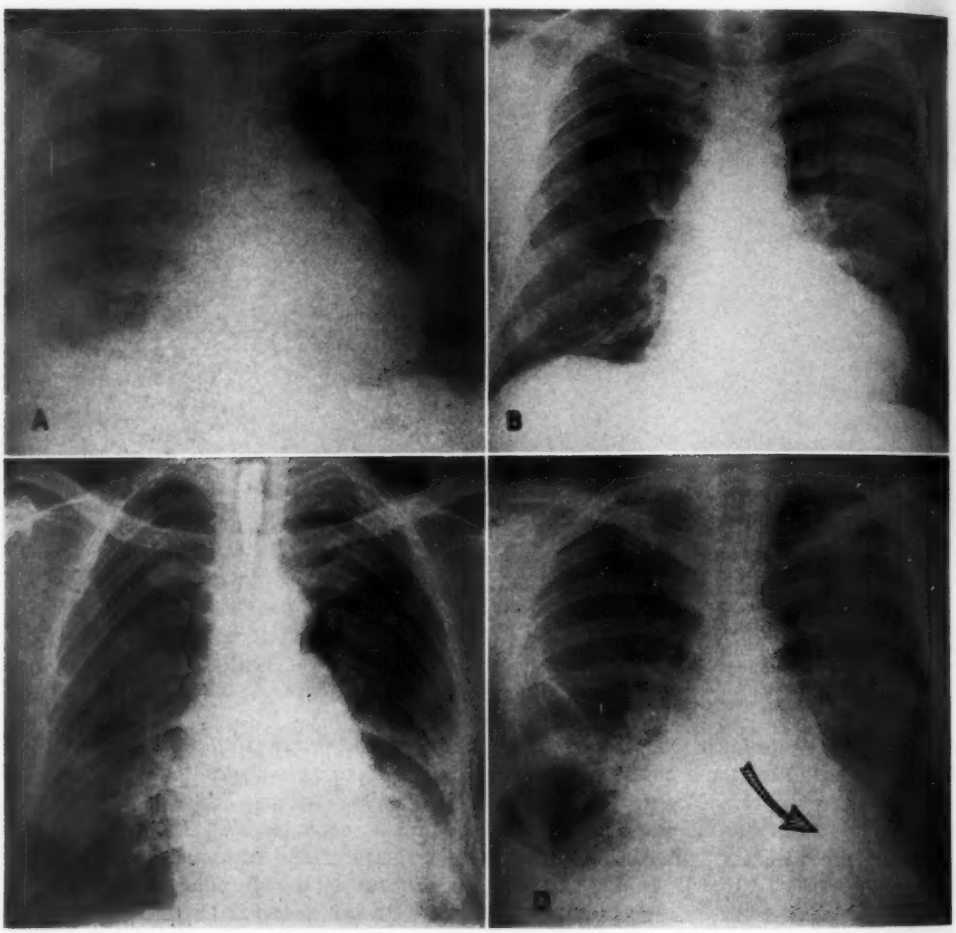

Fig. 4. The variable roentgen silhouette of constrictive pericarditis.
A. A 28-year-old man. Gross cardiac enlargement, prominence of the superior vena cava, and a right pleural effusion are evident.
B. A 41-year-old man. The heart is enlarged and the pulmonary vessels are engorged.
C. A 54-year-old man. Pulmonary engorgement is more marked, and the pulmonary artery segment prominent. Bilateral pleural thickening is apparent. No other cardiac lesions were present.
D. A 35-year-old man. Gross cardiac enlargement is associated with marked calcification in the atrioventricular groove (arrow). Fluid is apparent in the right pleural cavity. Note the superior vena caval prominence.

(*g*) *Fluoroscopy*. In about two-thirds of the cases, diminished pulsation, particularly of the left heart border, was described; in the remaining third, the pulsation was considered normal. Since the fluoroscopic examinations were made by a number of different radiologists, undue emphasis should not be placed on these observations.

(*h*) *Kymography*. Kymograms were obtained in 15 patients, in 4 of whom essentially normal pulsations of the cardiac borders were observed. In 11 instances there was altered pulsation of the left heart border. The commonest finding was a long, flat, diastolic plateau, associated with the "V" pattern, the 2 limbs of the "V" representing ventricular systole and diastolic filling respectively (Fig. 5). The amplitude of excursion was significantly decreased in most cases. In one of the patients who had excellent pulsations

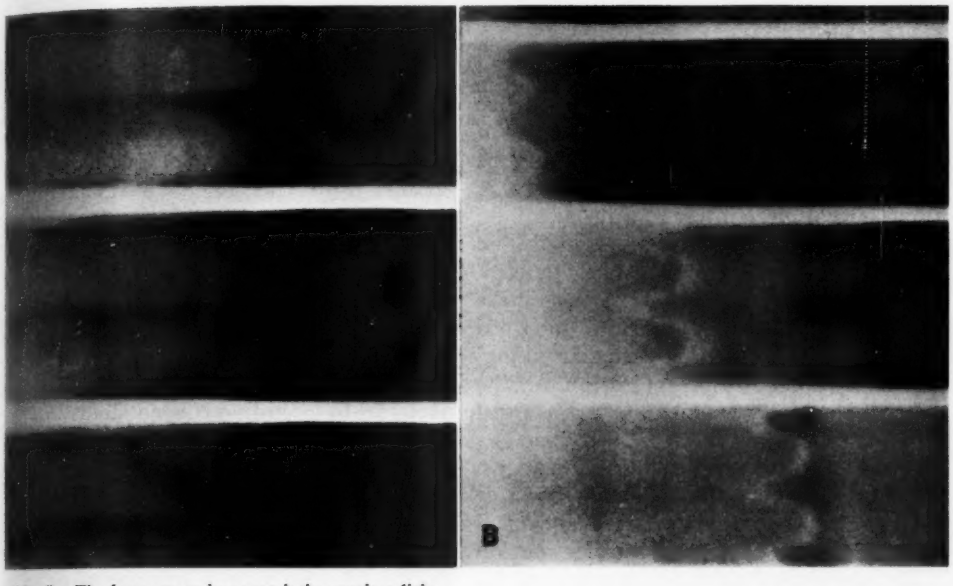

Fig. 5. The kymogram in constrictive pericarditis.
A. The diastolic lateral thrust ceases suddenly, and a long diastolic plateau ensues. This flat segment alternates with a "V," the lower limb of which represents the inward border movement of ventricular systole, while the upper limb represents early diastolic filling.
B. The kymographic recording of the calcified pericardium demonstrates much the same diastolic plateau as that noted in A.

at kymography, the pericardium was demonstrated at surgery to be thick and densely adherent to the myocardium.

(*i*) *Additional observations.* In 10 patients, pleural effusions were present: bilateral in 6, rightsided in 3, and leftsided in 1.

DISCUSSION

In 1937, Claude Beck described as the triad of chronic cardiac compression: (*a*) a small quiet heart, (*b*) high venous pressure, and (*c*) ascites (6). The myth that the heart in chronic constrictive pericarditis is "characteristically small and normal in outline" has been perpetuated by some even in the contemporary literature (5). The studies in this series demonstrate that there exists no such thing as a "characteristic" roentgen size or silhouette in chronic constrictive pericarditis. Instead, the heart size may range from normal to massive enlargement. Analysis of the chamber size demonstrates that the predominant enlargement is right ventricular, and a study of the pulmonary vascularity indicates that pulmonary engorgement, frequently accompanied by pleural effusion, is not uncommon. The prominence of the superior vena cava reflects the impedance of cardiac outflow, as does the azygos vein prominence.

The observation that cardiac enlargement is a relatively common accompaniment of constrictive pericarditis is by no means unique (8, 14, 18). The presence of cardiomegaly, should not discourage the diagnosis if the clinical data are sufficiently suggestive. The enlargement may not be proportionate to the degree of apparent right heart failure, in the sense that the hepatic enlargement, peripheral edema, and venous distention may seem incommensurate with the cardiac size. Pericardial calcification is a highly significant finding, in spite of the fact that extensive calcification in the absence of symptoms of pericardial compression has been observed.

The fluoroscopic and kymographic ob-

servations, indicating that normal cardiac pulsations are not inconsistent with the diagnosis of constrictive pericarditis, deserve comment. Fluoroscopic evidence alone might conceivably be ignored, in view of the highly subjective nature of the procedure. The normal kymographic studies are of more interest, since adequate objective base-line studies are available in patients without heart disease. The classical kymographic tracing of constrictive pericarditis shows an abrupt outward excursion during early ventricular filling, followed by an abrupt cessation of the lateral border movement and a prolonged flat segment during mid and late diastole. This reflects the inability of the ventricle to expand completely during diastole because of the compression of the thickened pericardium. The inward border motion at the end of diastole (systolic contraction) and the outward border motion at the end of systole (diastolic filling) form symmetrical limbs of a "V," alternating with the flat segments (Fig. 5). How is one to explain a kymogram with normal diastolic and systolic components in the presence of proved constrictive pericarditis? It can only be assumed that the particular segment of the cardiac silhouette studied by kymography in these cases was relatively unaffected dynamically by the constriction. Study of the surgical and pathologic material in this series, however, furnishes no adequate evidence to support this assumption, with a single exception. In this instance a patient was observed at surgery to have decreased right atrial and right ventricular pulsations with normal left ventricular pulsations. He responded well to freeing of the pericardium.

McKusick has suggested that compensatory increase in the amplitude of contraction occurs in areas where there is less impediment to ventricular motion (15). There was no physiologic evidence in any of the cases in this series to substantiate the concept that localized areas of constriction were present proximal to the ventricles or atria. This supports the conclusions of Sawyer, *et al.* (19), and Isaacs and his co-workers (12), that the major physiologic defect lies in the inability of the ventricles to fill normally during diastole.

The presence of apparent enlargement of the right cardiac chambers in association with the prominent pulmonary artery segment and large central pulmonary arteries may suggest the possibility of cor pulmonale on the basis of primary pulmonary parenchymal or vascular disease. The absence of an electrocardiographic pattern of right ventricular hypertrophy is useful in excluding this possibility.

That tuberculosis (4, 11, 18), traumatic hemopericardium (9), and purulent pericarditis (7) may cause constrictive pericarditis is well established. Rheumatic fever has been said not to be an etiologic factor (4, 18); yet a number of cases in which constrictive pericarditis has coexisted with rheumatic heart disease have been reported (11, 13, 16, 17), and some authors have expressed the belief that rheumatic fever may play a causative role (10, 17). This series included 2 additional cases of coexistent rheumatic valvular disease and chronic constrictive pericarditis. Both patients gave a history of migratory polyarthritis in childhood and 1 had suffered from chorea as well. The murmurs of mitral stenosis and insufficiency were heard in 1 case, and those of both mitral and aortic valvular involvement in the other.

Despite the weight of opinion and evidence against rheumatic fever as an etiologic factor in constrictive pericarditis, the question is by no means closed. This is particularly true in view of the failure to establish an etiologic agent in well over 50 per cent of cases (18).

SUMMARY AND CONCLUSIONS

Analysis of the radiologic findings in constrictive pericarditis demonstrated that there is no "characteristic" roentgen picture. In spite of frequent descriptions of a "small heart," the cardiac silhouette may appear enlarged in many cases, and

normal pulsation may be noted fluoroscopically and kymographically. Although pericardial calcification was demonstrated in only about half of the cases reported here, its presence is helpful when constrictive pericarditis is suspected. Commonly, the right ventricle, pulmonary artery segment, and the hilar pulmonary arteries are enlarged, and pulmonary engorgement is apparent.

If constrictive pericarditis is indicated clinically, such observations as cardiac enlargement, cardiac pulsations within the normal range, and radiologic evidence of pulmonary artery and right ventricular enlargement should not be considered incompatible with that diagnosis.

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SUMMARIO IN INTERLINGUA

Aspectos Radiologic de Operabile Morbo Cardiac. IV. Apparentia Variable de Pericarditis Constrictive

Le autores passa in revista 21 casos de pericarditis constrictive—omnes studiate extensemente tanto clinica- como etiam radiologicamente e omnes confirmate al operation. In 11 casos le historias e le constatationes pathologic lassava le etiologia dubitose. Signos e symptomas es discutite. Electrocardiogrammas preoperatori monstrava basse voltages in 7 casos, prolongation del intervallo P-R in 3, fibrillation auricular in 4, e alterationes del unda P in 2. Anormal undas T esseva notate frequentemente. Catheterisation cardiac demonstrava nulle significative gradientes de pression inter le venas cave e le atrio dextere, inter le atrio dextere e le ventriculo dextere, o inter le ventriculo dextere e le arteria pulmonar, con le exception de un patiente in qui, al operation, le presentia de stenosis del valvula pulmonic esseva constatate a parte le pericarditis constrictive.

Le analyse del constatationes roentgenologic in pericarditis constrictive demonstrava que il existe nulle characteristic configuration in ille medio. In despecto de

frequente descriptiones per altere autores del presentia de un parve corde, le silhouette cardiac pote apparer allargate in multe casos, e normal pulsation pote esser notate fluoroscopica- e kymographicamente. Le presentia de calcification pericardial es considerate como utile in le diagnose, ben que illo esseva demonstrabile in solmente 11 del casos in le presente serie. Communmente, le ventriculo dextere, le segmento de arteria pulmonar, e le hilar arterias pulmonar es allargate, e congestion pulmonar es apparente.

Si pericarditis constrictive es indicate super le base de considerationes clinic, le observation de phenomenos como allargamento cardiac, pulsationes cardiac intra latitudes normal, signos radiologic de allargamento de arteria pulmonar e de ventriculo dextere non deberea esser considerate como incompatibile con ille diagnose.

Scanographic Methods in Visualization of the Blood Vessels and Skeleton[1]

S. A. MORTON, M.D., and R. W. BYRNE, M.D.

NOW THAT surgical methods can be used to treat successfully lesions of the aorta and major blood vessels, it is of the utmost importance that the radiologist develop technics for visualization of the larger vessels of the body. When opaque material is injected into an artery, it diffuses with the blood and is rapidly carried away from the point of injection. If a single roentgenogram is made at the time of injection, one may obtain visualization of the opaque material, though usually in a rather limited area of the vessel.

If one uses a device for rapidly changing cassettes, a more extended view is obtained, showing the opaque material filling the vessels more peripherally. This method is usually quite adequate if one is dealing with the aorta or its iliac branches, but in investigation of the arteries of the leg, even a 14 × 17-inch film is not large enough to cover all the area desired. It was thought that the problem might be solved by a single exposure, using a 14 × 36-inch cassette, but while the bones of the entire leg were shown, the area of the vessels visualized by the opaque medium was still limited.

In a recent review of the subject, Rogoff discussed the various methods employed in angiography, presenting an excellent bibliography. He advocates a method using serial exposures on 14 × 36-inch films, and presents some excellent illustrations to exemplify this procedure. This, however, involves the use of a 14 × 36-inch Potter-Bucky diaphragm, which is not available in most departments and is a comparatively expensive item. We believe that scanographic methods will give accurate results with less expensive and more widely available equipment.

It seems exceedingly important to visualize the circulation of the entire leg. One must get an accurate idea of the extent of the runoff in the popliteal branches before undertaking to remedy a femoral block. Little will be gained by bridging a block if the circulation below is inadequate.

Astle and Wallace-Jones in 1953 described their method of femoral arteriography with the aid of a scanning device. Using their technic, we have consistently obtained excellent results. This method we now follow routinely and have even extended its use to other purposes. We believe simple scanographic equipment should be used more frequently and should be available in the x-ray departments of all hospitals where vascular surgery is being done.

Closely following the suggestions of Astle and Wallace-Jones, we have devised several small pieces of equipment. These include a 10-inch cone to the end of which is fastened a piece of lead 1/8 inch thick, with a narrow slit in it, 3 inches long and 1/8 inch wide, and a small hand-operated winch having a metal crank with a shaft 1/2 inch in diameter. A 14 × 36-inch cassette is used, loaded with two 14 × 17-inch films carefully butted end to end. The two films are used instead of a single 14 × 36-inch film because of ease of developing and convenience of storage of the finished radiograph.

For femoral arteriography the patient is placed on his back on the x-ray table. The 14 × 36-inch cassette is positioned under the leg so that its top is two finger breadths above the anterior-superior spine of the ilium. In the average patient the lower end of the film will then be just above the ankle (Fig. 1). The cone is at-

[1] From the Departments of Radiology, Columbia Hospital and Marquette University, Milwaukee, Wisc. Accepted for publication in January 1957.

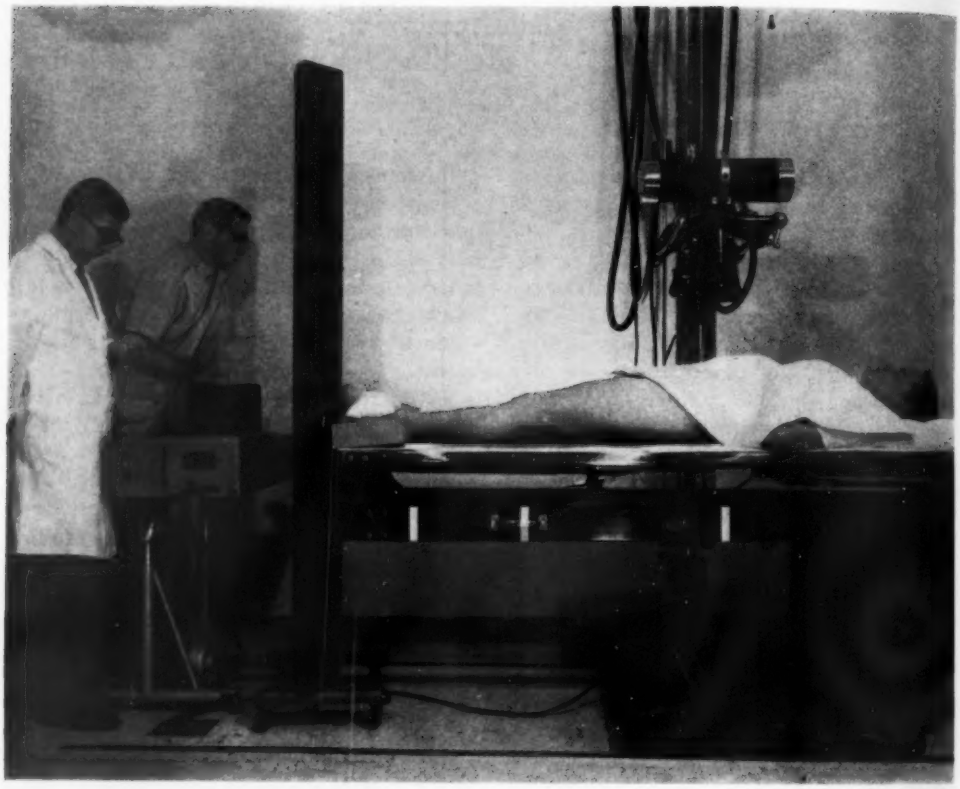

Fig. 1. Patient in position for femoral arteriogram, lying directly on a 14 × 36-inch cassette. Markings on the table edge are repeated on the floor for guidance of the winch operator.

tached to the tube so that the slit is at right angles to the long axis of the leg. The cord from the winch is tied to the tube stand. A focal-film distance of 30 inches is used and all locks are released so that the tube stand may travel freely.

Because the leg is a conical structure, with marked difference in thickness between the thigh and ankle, the speed with which the tube moves must vary with the part. To facilitate this, markers are placed on the floor showing when the tube is centered over the upper end of the cassette, when it is opposite the knee joint, and when it is opposite the ankle. These markers serve as a guide for regulating the speed at which the tube stand moves.

A stop watch is used to count off the seconds, and by watching the markers on the floor the operator of the winch knows the position of the tube in respect to the thickness of the leg and can vary the speed of travel accordingly. For an average leg an eight-second exposure is used, five seconds above the knee and three seconds below. After a few trial runs without x-rays, the operator of the winch is able to pull the tube stand from anterior-superior spine to ankle with a steadily increasing speed. The exposure factors vary somewhat with the size of the part, but the averages are 80 kvp, 25 ma, 30-inches distance. The timer is set for twelve to fifteen seconds.

The opaque medium used is 50 per cent Hypaque sodium, 30 c.c., which is injected into the femoral artery just below the inguinal ligament. The injection is made rapidly; it should not take more than five or six seconds.

The x-ray tube is placed 10 or 12 inches above the top of the cassette and, as the surgeon begins to inject the medium, the tube stand is started on its way toward the foot of the patient. The x-ray exposure is started and by the time 10 c.c. has been injected the tube should be at the top of the cassette. The stop watch is then set and the seconds counted aloud for the guidance of the winch operator. The tube progresses down the leg so that the band of radiation virtually follows the opaque medium as it progresses. It is thus possible to visualize the femoral artery and all its major branches (Fig. 2) with a single injection of opaque medium and with one exposure. The winch operator is protected by a lead screen.

Experience has taught that some variation of speed is necessary, depending on whether or not an arterial block is present and its location, and that there should be some difference in the amount of medium injected relative to the time of starting of the exposure. If it is felt that a rather marked obstruction is present, we wait until 15 c.c. of the contrast material has been introduced before the moving tube crosses the top of the plate. If there seems to be a lesser degree of obstruction, the exposure is started sooner, after injection of about 5 c.c. Because of the narrowness of the slit, no grid is needed and no film-changing device is used.

There is virtually no divergence of the rays in the sagittal plane, so that distances measured on the films are true. Therefore, in estimating the length of grafts that may be required, measurements may be made directly from the radiographs.

In order to interpret the arteriograms intelligently, the radiologist must have an accurate knowledge of the branches of the aorta and the iliac and femoral vessels.

Although the point of injection is below the inguinal ligament, there is sometimes enough reflux up the external iliac artery to fill its inferior epigastric and deep circumflex branches.

It is necessary to distinguish clearly between the femoral artery and its largest branch, the deep femoral artery. In cases of femoral occlusion this latter vessel enlarges and could be mistaken for the main femoral artery. The circumflex arteries and the perforating branches are often identified. One is impressed with the free anastomoses between the various branches of the femoral artery and about the knee joint. These features are beauti-

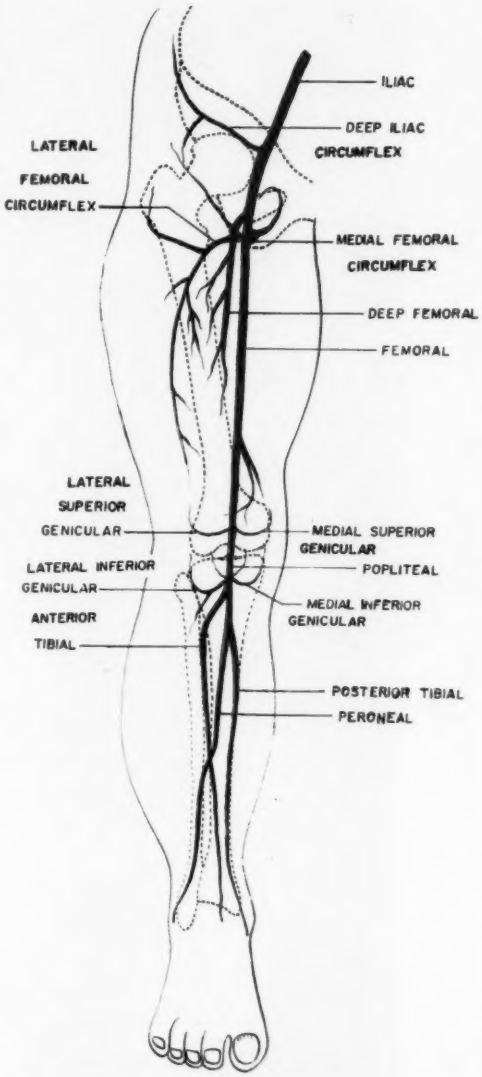

Fig. 2. Diagrammatic representation of the vessels seen in a normal femoral arteriogram.

Fig. 3. Normal femoral arteriogram. Note relatively small size of deep femoral vessel when main vessel is patent.

Fig. 4. A short complete femoral obstruction with adequate collateral anastomoses. Note good state of popliteal vessels.

Fig. 5. A complete high femoral obstruction with circulation to lower leg maintained through enlarged deep femoral vessel.

Fig. 6. Diffuse arterial disease. Multiple areas of narrowing and a poor "run-off."
Fig. 7. Long but localized lesion with good collaterals and a good "run-off."
Fig. 8. Same case as with Fig. 7, functioning graft.

Fig. 9. Simultaneous visualization of abdominal aorta, both iliacs, and both femoral vessels by scanographic technic.

fully displayed in the femoral aortograms, particularly if some degree of obstruction is present.

The popliteal artery, its branches about the knee joint, and its terminal branches, the anterior and posterior tibial vessels, are usually well shown. The peroneal branch of the posterior tibial artery is also usually outlined by the medium.

After a graft has been used, or the femoral vessel has perhaps been freed from extrinsic pressure or intraluminal plaques have been cleaned out, scanographic examination of the arterial system gives a graphic indication of the success or failure of the procedure.

Other uses of this procedure have been developed. It is possible to obtain visualization of the abdominal aorta, the iliac arteries, and the femoral vessels of both legs as far down as the knee. The injection is made with the patient lying on the abdomen as for an ordinary lumbar aortogram. The time factors differ from those for femoral arteriography. The tube travel is slower over the abdomen, particularly over the pelvis, but is speeded up considerably over the legs, so that when the tube reaches the mid thigh it is going about as fast as possible. Good plates have been obtained by using four seconds from the top of the plate to the greater trochanter and about three seconds for the part below. The speed of the tube must not be increased until the trochanters are passed. The voltage is from 90 to 95 kvp, and a longer slit is required to insure wide enough coverage of both legs. This slit is of the same width but is 4 3/4 inches long. Because of decreased secondary and scattered radiations, the shorter slit gives better detail, but satisfactory films can be made with the longer slit if the patient is not too large.

Since exact bone length can be measured on such films, we have used this same procedure to compare one leg with the other in orthopedic problems. The x-ray factors and the speed of tube motion are essentially the same as for femoral arteriography.

Radiographs of the entire spine can also be obtained by this method, which is sometimes of advantage in patients with scoliosis. Good plates showing the align-

ment of the vertebrae are produced without the necessity of an extra length Bucky diaphragm. There seems no reason why the method could not be adapted to examination of the spine in the upright position, though we have not tried this.

SUMMARY

A scanographic procedure based on the method of Astle and Wallace-Jones is described. By its use the arterial system of the entire lower extremity may be satisfactorily demonstrated with one injection and a single x-ray exposure.

Other uses of this procedure include leg length measurement, combined visualization of the lumbar aortic and bilateral femoral system of vessels, and demonstration of the entire spine.

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SUMMARIO IN INTERLINGUA

Methodos Scansiographic in le Visualisation del Vasos Sanguinee e del Skeleto

Es describite un technica scansiographic pro le demonstration del systema arterial del gamba integre, basate super le methodo del Astle e Wallace-Jones. Es usate duo pelliculas de 14 × 17 pollices in un sol cassetta de 14 × 36 pollices.

Le cassetta es placiate sub le gamba del patiente qui jace in position dorsal super le tabula Roentgen, e le porta-tubos es movite per medio de un manivella a operation manual al longo del gamba integre, con variation del rapiditate de accordo con le spissitate del differente partes. Es requirite non plus que un sol injection de substantia de contrasto e un sol exposition a radios X.

Altere usos del technica es le mesuration del longor de gambas, le visualisation combinate del systema vascular lumbar, aortic, e femoral bilateral, e le demonstration del longor total del columna vertebral.

Correlation of Anatomic and Roentgen Changes in Arteriosclerosis and Syphilis of the Ascending Aorta

GWILYM S. LODWICK, M.D., and WILLIAM S. GLADSTONE, M.D.

CALCIFICATION of the intima of the ascending arch of the aorta has long been recognized by radiologists as evidence of syphilitic aortitis. Jackman and Lubert (1) in 1945 reported the results of a comparison of chest roentgenograms from 66 autopsied cases of syphilitic aortitis and 62 autopsied cases of severe atherosclerosis of the ascending aorta. They found roentgen evidence of calcification in the ascending aorta in 22.7 per cent of the cases of syphilis as compared to 3.2 per cent of the cases of atherosclerosis. Leighton (2), having studied a series of patients whose chest roentgenograms demonstrated calcification of the ascending aorta, reported that 66 per cent showed positive serologic evidence of syphilis. Others have made similar observations (3). Intimal calcification not thus accounted for is due either to clinically unrecognized syphilis in individuals in whom the serologic evidence has become negative or "burned out," or to calcific atherosclerosis resulting from arteriosclerosis.

While the radiologist correlates calcification of the ascending aorta with syphilitic aortitis, the pathologist commonly discovers calcified atheromatous plaques in the root or ascending arch of the aorta and relates these findings to arteriosclerosis. In syphilitic aortitis, the expected pathologic changes are elongation and dilatation of the aorta and wrinkling and fissuring of the intima. In an effort to reconcile the apparently divergent points of view of the radiologist and the pathologist, we have undertaken a comparative investigation of the roentgen changes of atherosclerosis of the aorta and syphilitic aortitis. These studies include radiography and measurement of gross autopsy specimens from patients with arteriosclerosis and syphilis, as well as measurement of the aortic shadow in chest roentgenograms of patients with visible calcification in the ascending arch in both conditions. From the results of this study, we conclude that the appearance and distribution of intimal calcific plaques in the ascending aorta for the two diseases are so distinct, and differences in length and width of the aortic arch sufficiently great, that a roentgen distinction may be made in at least 90 per cent of cases.

STUDY OF GROSS AUTOPSY SPECIMENS

For reliable determination of the location and character of calcific atheromatous intimal deposits, roentgenograms were made of 72 aortas obtained at autopsy. In each instance, the specimen consisted of the entire ascending aorta, the arch, and a segment of descending aorta of variable length. The heart and aortic valve were radiographed with the aorta in most instances. The roentgenograms were obtained *en face* rather than *en profile*.

The patients from whom the specimens were obtained ranged in age from nineteen to eighty-four years. All were males from a general medical and surgical Veterans Administration Hospital. Seven patients were under thirty years of age; 6 of these had no evidence of intimal calcification in the aorta. Evidence of calcification was lacking in 10 of 13 patients under fifty years and in 1 man of sixty.

Serologic tests for syphilis were positive in 2 patients, but in these neither clinical nor autopsy findings demonstrated evidence of syphilitic aortitis.

The roentgen shadow of each aorta was divided into 4 divisions by boundaries

[1] Accepted for publication in January 1957.
[2] Professor and Chairman, Department of Radiology, School of Medicine, University of Missouri.
[3] Now in private practice in Kalamazoo, Mich.

which are arbitrary, but designed to separate areas having a high incidence of intimal calcification from areas with little calcification (Fig. 1). The first division, or Part I, comprises the first 2 cm. of the aorta above the valve, or the root of the vessel. The lower part of this segment is surrounded by ventricular musculature, and the upper part is covered by the reflection of the pericardium. Part II extends from the upper margin of Part I to a point 1 cm. proximal to the junction of the innominate artery with the aorta. This part is divided equally into medial (Part II-M) and lateral (Part II-L) halves, because of variations in the amount of calcification appearing in these two areas, and because of differences in visibility in the chest roentgenogram. The posteromedial wall or lesser curvature of this ascending segment, II-M, lies in contact with the pulmonary conus and right pulmonary artery, which passes behind the aorta from left to right. These structures splint this segment of the aorta, which is much shorter than the anterolateral wall or greater curvature. The lateral segment by contrast is free of contact with firm supporting structures. In the routine postero-anterior projection of the chest, the shadow of II-M lies over the spine and is obscured by it, while II-L is observed just to the right of the spine, highlighted against the radiolucent background of lung, especially when the aorta is elongated and more laterally located.

Part III begins at the upper margin of Part II, and terminates 1 cm. distal to the junction of the left subclavian artery with the aortic arch. Portions of this segment lie in contact with the trachea and pulmonary vessels, and in addition receive support from the three major arteries arising from the greater curvature. Part IV is the descending thoracic aorta, beginning at the most distal margin of Part III, and terminating at the end of the specimen. It is immobilized in part by the intercostal arteries, the spine, and, in its upper portion, the pulmonary vessels of the hilus.

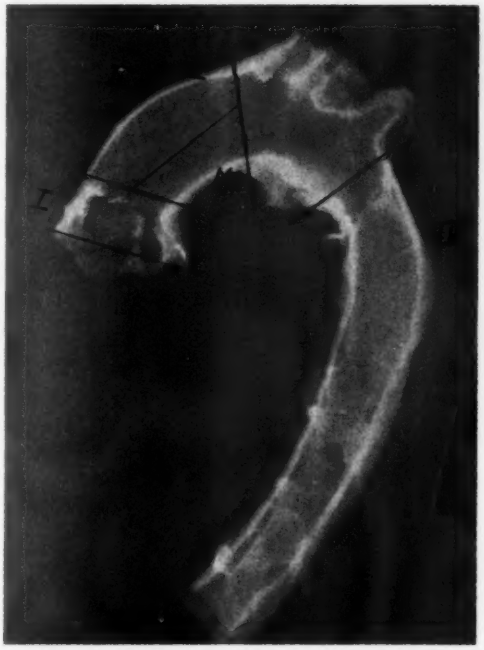

Fig. 1. Roentgenogram of aorta, showing the four parts and the medial and lateral subdivisions of Part II.

In the 61 aortas in which intimal calcification was detected, the distribution was as follows: Part I, 42 cases; Part II, 12 cases; Part III, 54 cases; Part IV, 56 cases.

In Part I the incidence of calcification was 68.8 per cent. Most commonly the calcific plaques were found to be located around the ostia of the coronary arteries and on the wall above the valve attachments. There was a high incidence of associated coronary artery calcification.

The incidence of calcification in Part II was 19.6 per cent. In this section calcification was usually in the form of isolated plaques. These were located in 7 instances in II-M alone, in 3 instances multiple deposits occurred in both II-M and II-L, and in 2 instances isolated plaques crossed the borderline to lie in both II-M and II-L. In only 3 of the 12 specimens with calcification was the plaque located sufficiently laterally to project against the lung field in the postero-anterior film (4.9 per cent), while in only 1 instance (1.6

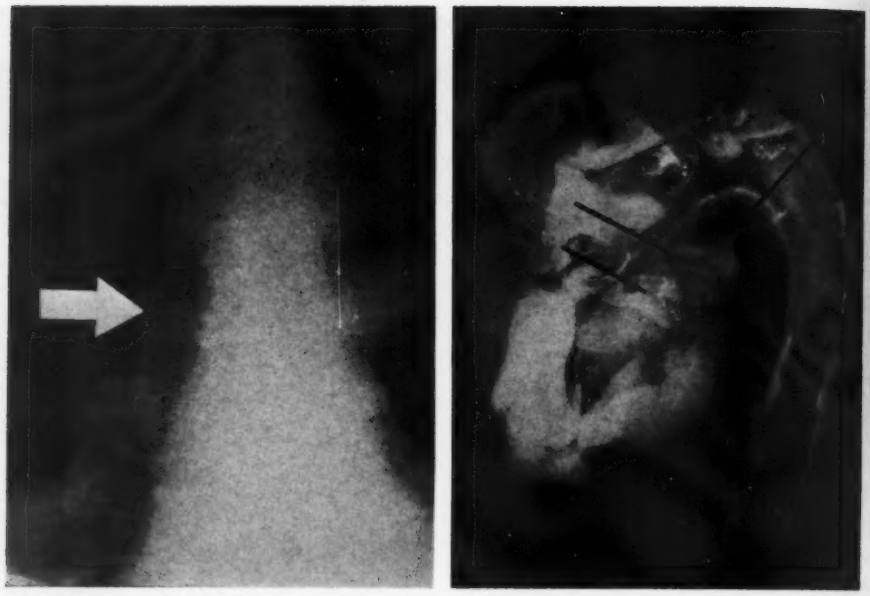

Fig. 2. Large calcified atheroma in Part II-L, demonstrated in chest roentgenogram. Atheromatous plaques occur more frequently in Part II-M, but are hidden by the spine.

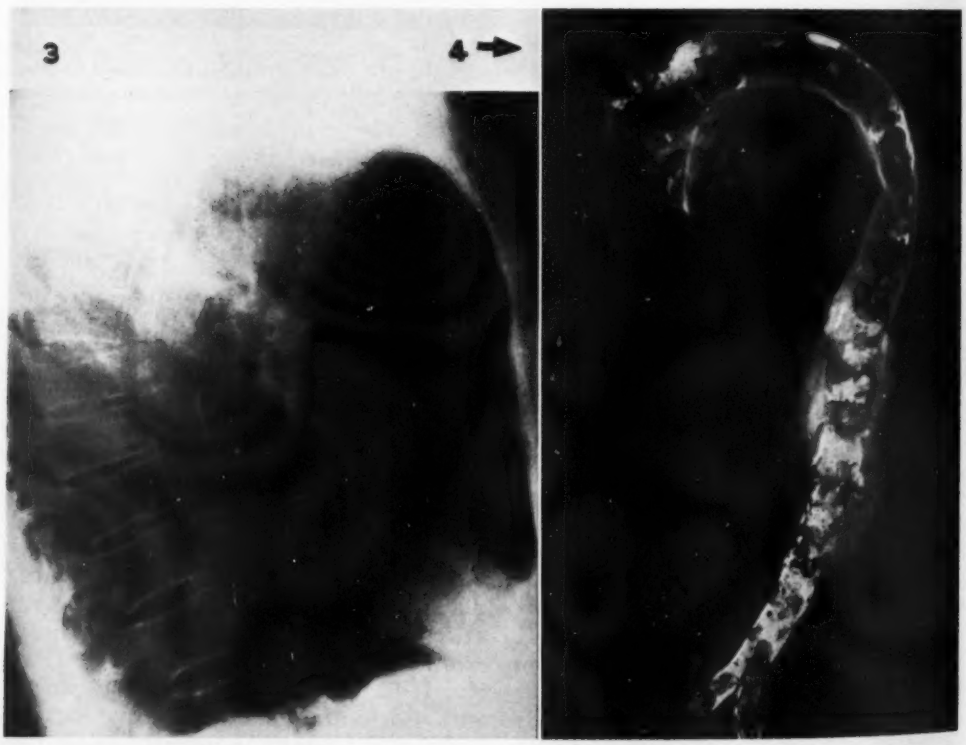

Fig. 3. Typical distribution of atheromatous intimal calcification in arteriosclerosis. There is a large plaque in Part II-M, but Part II-L is free of visible calcium.

Fig. 4. Typical distribution of atheromatous intimal calcification in arteriosclerosis. Roentgenogram of specimen.

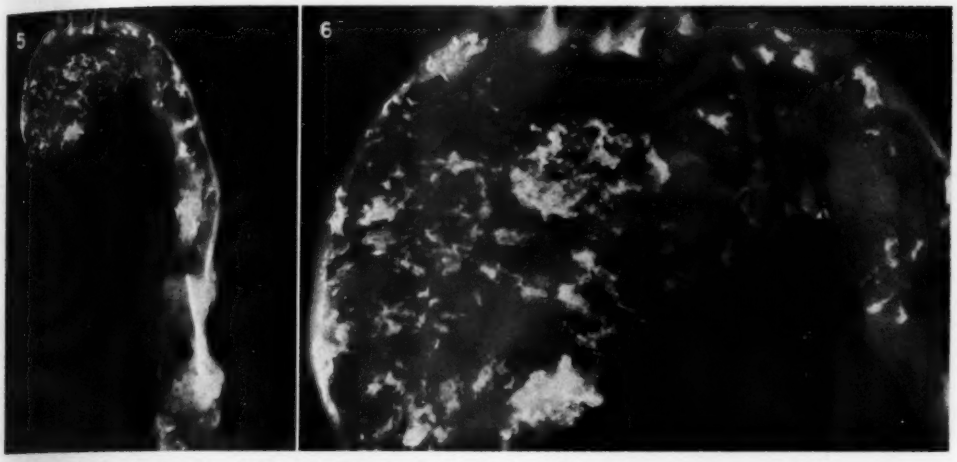

Fig. 5. Typical distribution of intimal calcification in syphilis. Roentgenogram of specimen.
Fig. 6. Enlargement of Fig. 5, showing small, flaky calcification in the ascending portion, typical of syphilitic aortitis. Compare with Fig. 4.

per cent) was the calcification actually detectable in the clinical roentgenogram (Fig. 2).

Calcification was present in Part III in 88.5 per cent of the cases. It was observed principally around the ostia of the great vessels emanating from the arch. In Part IV the incidence was 91.8 per cent. The deposits occurred as isolated plaques and around the ostia of the intercostal arteries.

To summarize, it was found that calcification is frequently observed in the ascending aorta in atherosclerosis without underlying syphilitic aortitis. However, because the calcification is located predominantly in the root and posteromedial wall of the aorta, zones obscured by the density of the spine, it is only rarely visualized in the postero-anterior chest film.

COMPARISON OF DISTRIBUTION AND APPEARANCE OF INTIMAL CALCIFICATION IN ATHEROSCLEROSIS AND IN SYPHILITIC AORTITIS

Figures 3 and 4 demonstrate especially well the distribution and appearance of atheromatous intimal calcification in arteriosclerosis. The lateral chest film reproduced in Figure 3 typifies our autopsy observations: heavy calcification in the root and coronary ostia, isolated plaques in the posteromedial wall of Part II (not visible in the postero-anterior projection), calcified plaques around the ostia of great vessels of the arch, and most extensive calcification in the descending aorta. In Part IV the heaviest calcification is seen on the wall nearest the spine and nearest the hilus. The roentgenogram of the specimen (Fig. 4) also demonstrates the typical distribution of intimal calcification in atherosclerosis, the greatest amount being in the abdominal portion of the aorta. In addition, the calcific plaques tend to be large, dense, angular, and isolated. Roentgenograms of specimens from patients with known syphilitic aortitis (Figs. 5 and 6) demonstrate a very different distribution and pattern of intimal calcification. Paralleling the anatomic distribution of the typical involvement of the aorta in syphilis, the greatest volume of calcification is found in the ascending aorta and arch, where it is seen as innumerable angular, flaky plaques, smaller than those in atherosclerosis. Both the medial and lateral halves of Part II are extensively calcified in syphilis. The patterns of calcification in these two conditions are so strikingly different that on this basis alone it is possible to distinguish between

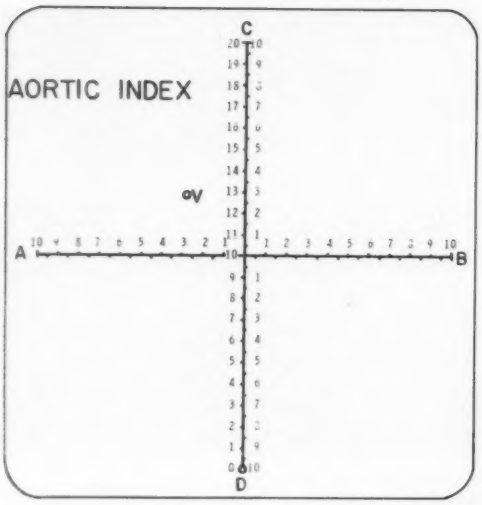

Fig. 7. Transparent overlay used for determining aortic index. When the overlay is properly centered over the cardiac shadow, "V" is approximately at the aortic valve.

atherosclerosis and syphilitic aortitis from the roentgenogram of the gross specimen.

METHOD OF DETERMINING ELONGATION OF THE AORTA

For an adequate comparison of the clinical roentgenograms of patients with and without visible calcification in the ascending aorta, a method of determining aortic length is required. The most accurate technic of measuring the dimensions of the aorta involves the use of aortography (4). This procedure must be done in the living patient, because of the known discrepancies between measurements in the living and in the dead (5). Determination of the length of the aorta, however, is rarely an important enough diagnostic procedure to warrant the risks of aortography. The transverse measurement of the arch alone does not provide sufficiently valid data.

We have developed a technic for measuring the height of the aortic arch by taking the location of the pathologically calcified aortic valve as an indication of the proximal end of the aorta. Using a simply constructed transparent plastic overlay with crosslines for centering and aligning (Fig. 7), we have found, through study of roentgenograms demonstrating calcified aortic valves, that the location of the valve (Point V on the overlay) bears an approximate relationship to the cross lines. It is acknowledged that the location of the aortic valve as thus determined is only a rough approximation. The cardiac variations which introduce error are principally as follows:

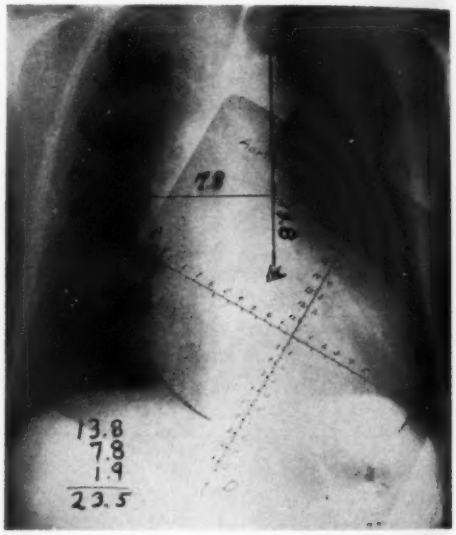

Fig. 8. Proper centering of transparent overlay on the cardiac silhouette.

1. *Heart size.* As used, point V has a fixed relationship to the center of the cardiac silhouette. Actually, the relationship of V to the center should vary with heart size, but the extent of the error is small, and its correction would complicate measurement.

2. *Heart position.* Variations introduced by different degrees of horizontal and vertical positioning of the heart shadow are largely corrected by alignment of the AB axis with the longest cardiac axis. Rotation of the heart on the transverse axis introduces a small error, difficult to correct.

3. *Two-dimensional measurement.* Introduction of a third dimension through use of the lateral projection of the chest

is a refinement which should increase the accuracy of measurement. While the technic of measurement would be only slightly more complicated, the selection of two-dimensional measurement is necessitated by lack of lateral chest roentgenograms for many of the cases available to us for study.

To measure the aorta, the overlay is placed over the shadow of the cardiac silhouette so that the AB axis is aligned with the greatest cardiac dimension. The overlay is adjusted until the numbers on each axis are identical, and the cross lines are at the center of the cardic silhouette. The hole V is now at the estimated site of the aortic valve, and the spot is marked with a wax pencil. The highest and broadest points on the aortic arch are marked on the film, and a vertical reference line is drawn from the highest point downward (Fig. 8). Again using the AB and CD axes, the height and width of the aortic shadow are determined. These measurements, when totaled, constitute the aortic index.

"Normal" distribution curves for aortic indices of males and females over twenty years of age have been constructed from data collected from 228 subjects without clinical evidence of syphilitic aortitis. For males, the distribution curve is of the expected contour, the extremes measuring 12.4 cm. and 24.7 cm.[2] respectively (Fig. 9). The average index for all ages is 18.5 cm. Less than 10 per cent of the indices exceed 22 cm. Average indices for males by decade are shown in Figure 10.

For females, the extremes of the distribution curve measure 12.4 cm. and 19.8

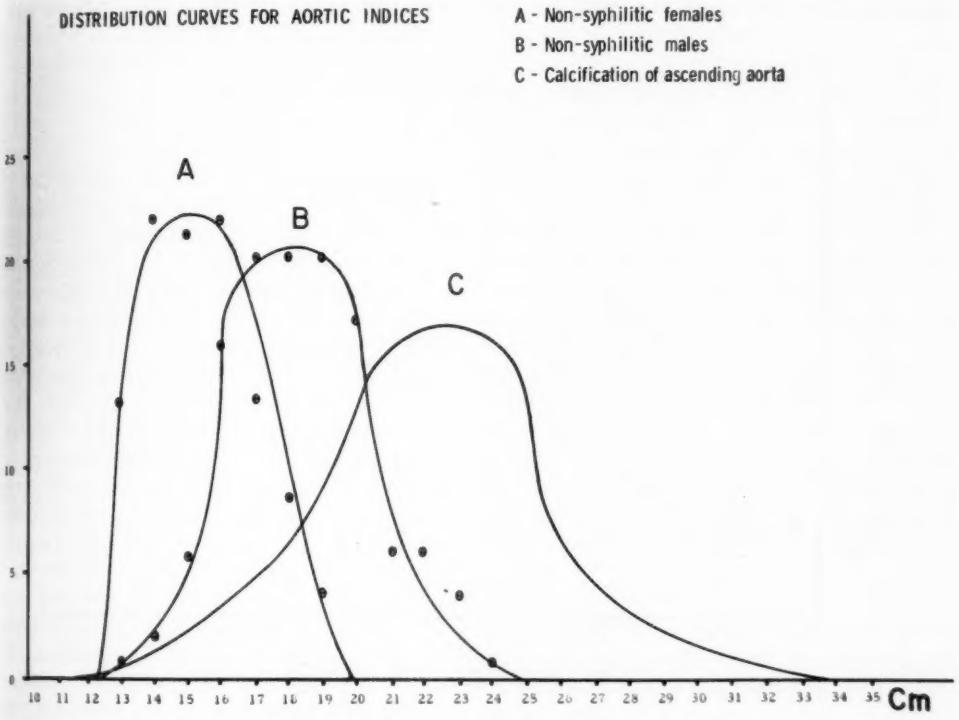

Fig. 9. Distribution curves for aortic indices.

[2] This high figure was obtained in a sixty-five year-old man with unusually severe arteriosclerosis of peripheral vessels and spontaneous sloughing of one foot.

cm. respectively (Fig. 9). The average index for all ages is 15.0 cm., and less than 10 per cent of indices are greater than 18.5 cm. The average indices, by decades are shown in Figure 10.

CALCIFICATION OF THE INTIMA OF THE ASCENDING AORTA IN CLINICAL ROENTGENOGRAMS

Postero-anterior chest roentgenograms which unequivocally demonstrate calcification of Part II-L of the ascending aorta were available in 62 cases. Data from serologic tests for syphilis were available for 61 patients: 52 males and 9 females. The tests were positive in 43 males and in 6 females, an overall incidence of 80.3 per cent. The patients with negative tests (19.7 per cent) are assumed to have arteriosclerotic calcification, although late syphilis with "burned out" serology cannot be excluded.

Aortic indices were determined for the 62 cases showing calcification in Part II-L. The average was 22.4 cm., with a range from 11.5 to 33.9 cm. The distribution curve for these cases is shown in Figure 9(C). In comparing the distributions plotted on this chart, we find that approximately 79 per cent of all patients with calcification in the ascending aorta have aortic indices greater than 20.0 cm. By contrast, approximately 79 per cent of all cases represented by the distribution curve B, for non-syphilitic males, have aortic indices of 20.0 cm. or less, while 100 per cent of females of the sample from which the distribution curve A was constructed have aortic indices of 20.0 cm. or less.

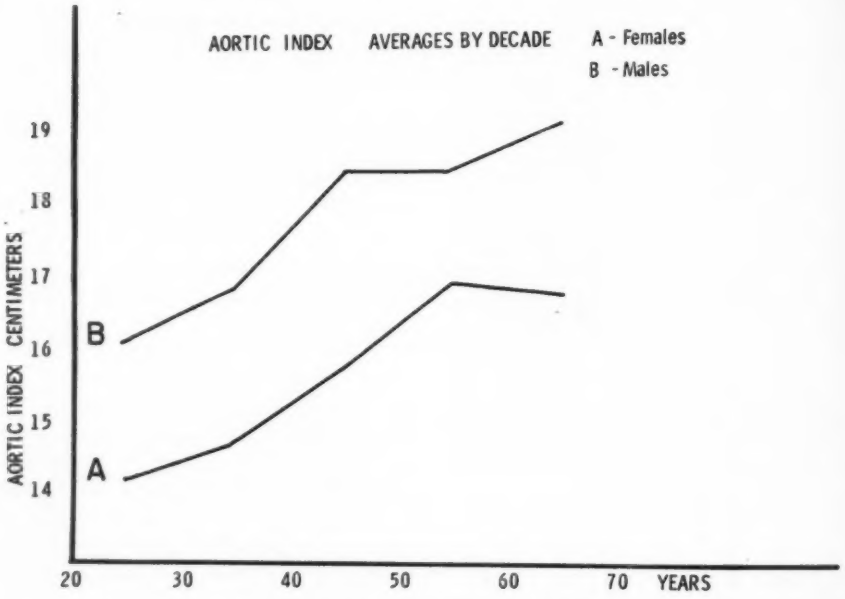

Fig. 10. Aortic index averages by decades.

When serologic tests for syphilis are considered in relation to aortic indices in those cases with calcification of the ascending aorta, it is found that none of the females with negative tests have indices greater than 20.0 cm., while in none of the men with negative serology does the index exceed 24.2 cm. Taking both sexes together, 85.1 per cent of all cases with aortic indices greater than 20.0 cm. and 100 per cent of all cases with indices in excess of 24.2 cm. showed positive sero-

logic evidence of syphilis. It should be noted, however, that in the "normal" distribution curve for males, aortic indices do occasionally exceed this figure (1–2 per cent).

To summarize our experience, positive serologic evidence of syphilis is found in 4 out of 5 cases with roentgen evidence of calcification in the ascending aorta. When the degree of elongation and tortuosity of the aorta, as measured by the aortic index, is considered along with the presence of calcification, the probability of syphilis increases to 9 or more times out of 10.

DISCUSSION

Infrequent occurrence of atheromatous calcification in the greater curvature portion of the ascending aorta probably can be explained by results of other studies. Generally, atherosclerosis is known to occur less frequently in vessels that are subject to stretching, bending, and massage, such as the large arteries at the knee and elbow (6). The same is true of segments of the aorta where pulsation of the walls is not limited by supporting or overlying tissues (7). This freedom to pulsate without extrinsic limitation is noticeably present in the ascending arch, where the only limiting structures are at the base, the posteromedial wall, and the attachment of the great vessels of the neck.

In addition, the aorta suffers loss of elasticity with aging (8, 9). This loss occurs earlier and more rapidly in portions of the wall which have limited excursion because of attachment of supporting structures. Localized atheromatous changes develop in these same areas, particularly where the aorta lies in contact with the spine, and at the bifurcation. It is believed that the tendency for atherosclerosis to develop around the ostia of the large vessels of the arch and the intercostal arteries is due to stiffening of the wall by these structures (7).

From these findings, we would assume that lack of fixation in Part II-L of the ascending aorta might well account for the relative absence of atherosclerotic calcification in that part.

This immobilization theory has been applied to explain the presence of intimal atherosclerosis in syphilitic aortitis. Syphilis causes extensive disorganization and destruction of the muscular layers and elastic lamellae of the media, to be followed by secondary degenerative changes in the intima. During the destructive phase, the weakened aortic wall is stretched in both circular and longitudinal dimensions, resulting in diffuse dilatation and elongation of the vessel. In the healing phase, the damaged muscular and elastic elements are replaced by dense inelastic collagenous tissue, with impairment of the pliability of the aortic wall. This stiffening is believed to promote atheromatous change (10). Since aortitis is diffuse, the calcification is also diffuse; in the chest film it appears to be more continuous than atherosclerotic calcification, which usually is discontinuous.

It appears that calcification in syphilitic aortitis is evidence of a healed, inactive phase of the disease. We have observed continued enlargement of the aorta in cases of active syphilitic aortitis but, once calcification of the intima is established, further enlargement has not occurred.

SUMMARY

The following conclusions are based upon gross anatomic and roentgen studies of normal, atherosclerotic, and syphilitic aortas.

1. Calcified atheromatous intimal plaques are not uncommonly found in the ascending aortic arch in the absence of syphilitic aortitis, but they are rarely observed in the postero-anterior chest roentgenogram because their posteromedial location is overshadowed by the spine. The anterolateral wall of the ascending aorta is not a common site for calcified atheromatous plaques.

2. By contrast, intimal calcification in syphilitic aortitis is found in the entire circumference of the ascending aorta. The increased length of the syphilitic

aorta displaces the ascending aorta to the right, where calcific changes can be better demonstrated against the radiolucent lung background.

3. A simple technic is presented for estimating the degree of elongation and tortuosity of the aorta (aortic index). Distribution curves are plotted for the aortic indices of "normal" non-syphilitic males and females.

4. Limiting our study of intimal calcification to cases in which roentgenograms demonstrate calcification in Part II-L, we find that 80 per cent of such cases have positive serologic evidence of syphilis. The aortic index, used as an additional screening factor, improves the correlation with proved syphilis to 90 per cent or better.

NOTE: The research on aortas was conducted at the Veterans Administration Hospital, Iowa City, Iowa, and was supported in part by research funds granted by the Veterans Administration. The clinical material comes from the Radiological Service, Veterans Administration Hospital, Iowa City, the Department of Radiology, University Hospitals, Iowa City, and the Department of Radiology, Medical Center of the University of Missouri, Columbia, Mo.

We are indebted to Dr. Kenneth Cross of the Laboratory Service, Veterans Administration Hospitals, Iowa City, and to Dr. Emory Warner of the Department of Pathology, University Hospitals, Iowa City, for permitting us use of material which made this study possible.

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SUMMARIO IN INTERLINGUA

Correlation del Alterationes Anatomic e Roentgenologic in Arterosclerosis e Syphilis del Aorta Ascendente

Esseva effectuate un studio comparative del alterationes roentgenologic in atherosclerosis del aorta e 61 casos de aortitis syphilitic.

Calcific placas atheromatose intimal esseva trovate non incommunmente in le ascendente arco aortic in le absentia de aortitis syphilitic, sed illos occurreva rarmente in pariete aortic antero-lateral. Per contrasto, calcific placas intimal in aortitis syphilitic esseva trovate in le integre circumferentia del aorta ascendente.

Esseva etiam effectuate determinationes del longor e largor del aorta per le simple medio de un transparente scala de plastico. Assi un indice aortic (i.e. le summa del longor e del largor del aorta) esseva establite. In un alte procentage del casos, il esseva trovate que un indice de plus que 20,0 cm correspondeva a positive tests serologic pro syphilis.

Super le base del resultatos de lor studio le autores conclude que le differentias del apparentia e distribution del calcific placas intimal in le aorta ascendente e etiam le differentias del longor e largor del arco aortic es si marcate in le duo morbos, que lor differentiation roentgenographic es possibile in al minus 90 pro cento del casos.

Half-Value Depth and Fall-Off Ratio as Functions of Portal Area, Target-Skin Distance, and Half-Value Layer[1]

WILLIAM J. TUDDENHAM, M.D.[2]

THE PURPOSE of this communication is to present, in convenient form, values for the half-value depths and fall-off ratios corresponding to the entire range of radiation qualities, portal areas, and target-skin distances likely to be encountered in clinical roentgen therapy. Compilation of these data is believed to be useful for several reasons. *First*, these parameters have proved extremely useful in specifying superficial radiation beams, and it is thought that a graphic comparison of the data will facilitate treatment planning for superficial lesions. *Second*, though these values have seldom been considered in the planning of intermediate or deep therapy, they do provide, in some measure at least, an index of the distribution of radiation of higher energy in tissue. It is believed, therefore, that the data will enable the student to visualize readily the effect of changing kilovoltage, external filter, portal size, and target-skin distance on the distribution of radiation within a patient's tissues. *Third*, interest in the calculation of the integral dose (7) delivered in roentgen diagnosis and therapy is increasing, because of its apparent correlation with the systemic effects of irradiation. Among the factors required for the calculation of the integral dose is the half-value depth of the incident radiation. The data presented here should therefore be of aid in this computation.

DEFINITIONS

Half-value depth, or D1/2, is the depth in tissue at which the radiation dose equals 50 per cent of the surface dose. It is thus a clinical expression of the penetrating power of a specified radiation beam. Similarly, D9/10 is the depth in tissue at which the radiation dose equals 10 per cent of the surface dose. Fall-off ratio, or F.O.R., is defined as the D9/10 of a specified radiation beam divided by its D1/2. The fall-off ratio is, therefore, a clinical expression of the absorption rate of the specified radiation in tissue.

THE DATA

1. Range of Data: Data are presented for half-value layers of 1 to 8 mm. Al and 0.4 to 15 mm. Cu, for portal areas varying from 5 to 400 sq. cm., and for target-skin distances commonly employed in this clinic. In addition, data applying specifically to the Philips and Chaoul contact-therapy units, and to a beryllium-window x-ray tube (Machlett O.E.G.-60) superficial-therapy unit are presented for half-value layers, portal areas, and target-skin distances appropriate to these generators.

2. Sources of Data: The data presented here for half-value layers of 1 mm. Al through 4 mm. Cu are all based on depth-dose tables published in *Physical Foundations of Radiology* (13). These tables were chosen as a basis because of their almost universal acceptance in clinical roentgen therapy in America. Moreover, repeated careful comparisons with other published data (1, 3) indicate that, at least between half-value layers of 1 and 8 mm. Al, the figures represent mean values of all the available measurements.

The selection of a basis for the data pertaining to the contact- and superficial-therapy units was much more difficult because of the relatively poor agreement among the several studies which have been reported in this field (2, 4, 8, 11, 12, 14, and 15). This lack of agreement is

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TABLE I: D1/2 AND FALL-OFF RATIO AS FUNCTIONS OF H.V.L., T.S.D., AND PORTAL AREA

h.v.l. mm.	T.S.D. cm.	Cone Diam. cm.	Area cm.^{2}	D1/2 cm.	D9/10 cm.	Fall-off Ratio
		Philips Superficial Therapy Unit: 45 kv				
0.30 mm. Al	2.0	1.0	0.79	0.25	1.2	4.8
		2.0	3.14	0.28	1.3	4.6
	4.0	1.0	0.79	0.40	1.8	4.5
		4.5	15.9	0.42	1.9	4.5
1.15 mm. Al	2.0	1.0	0.79	0.44	1.9	4.3
		2.0	3.14	0.48	2.0	4.2
	4.0	1.0	0.79	0.62	2.5	4.1
		4.5	15.9	0.73	2.9	4.0
1.75 mm. Al	2.0	1.0	0.79	0.50	2.2	4.4
		2.0	3.14	0.57	2.4	4.2
	4.0	1.0	0.79	0.78	3.1	4.0
		4.5	15.9	0.90	3.4	3.8
		Chaoul Superficial Therapy Unit: 60 kv				
3.3 mm. Al	1.5	1.5	1.77	0.30	2.3	7.7
		2.5	4.92	0.40	2.7	6.7
	3.0	2.0	3.14	0.87	3.7	4.3
		2.5	4.92	0.92	3.7	4.0
	5.0	2.0	3.14	1.0	4.1	4.1
		2.5	4.92	1.2	4.3	3.6
		3.0	7.10	1.3	4.7	3.6
		4.5	15.9	1.4	4.9	3.5
		Machlett O.E.G. 60 Beryllium-Window Tube Unit: 50 kv				
(Such units can be operated at voltages ranging from 2 to 50 kv. It has been found most satisfactory in this clinic, however, to operate at 50 kv in all cases and vary only the external filter to obtain the desired half-value layer)						
0.07 mm. Al	10	1.5	1.8	0.11		5.6
	15	5	20	0.11		5.6
	20	10	80	0.11		5.6
0.14 mm. Al	10	1.5	1.8	0.25		5.7
		5	20	0.25		5.7
		10	80	0.25		5.7
	15	1.5	1.8	0.26		5.7
		5	20	0.26		5.7
		10	80	0.26		5.7
	20	1.5	1.8	0.27		5.7
		5	20	0.27		5.7
		10	80	0.27		5.7
0.43 mm. Al	10	1.5	1.8	0.52		5.4
		5	20	0.53		5.4
		10	80	0.54		5.4
	15	1.5	1.8	0.54		5.4
		5	20	0.55		5.4
		10	80	0.56		5.4
	20	1.5	1.8	0.55		5.4
		5	20	0.57		5.4
		10	80	0.58		5.4
0.87 mm. Al	10	1.5	1.8	1.0		4.0
		5	20	1.1		4.0
		10	80	1.1		4.0
	15	1.5	1.8	1.1		4.0
		5	20	1.2		4.0
		10	80	1.2		4.0
	20	1.5	1.8	1.2		4.0
		5	20	1.2		4.0
		10	80	1.3		4.0

TABLE I: D1/2 AND FALL-OFF RATIO AS FUNCTIONS OF H.V.L., T.S.D., AND PORTAL AREA—*Cont.*

h.v.l. mm.	T.S.D. cm.	Cone Diam. cm.	Area cm.²	D1/2 cm.	D9/10 cm.	Fall-Off Ratio
		Machlett O. E. G. 60 Beryllium-Window Tube Unit: 50 kv				
1.8 mm. Al	10	1.5	1.8	1.4		3.8
		5	20	1.6		3.8
		10	80	1.7		3.8
	15	1.5	1.8	1.6		3.8
		5	20	1.7		3.8
		10	80	1.9		3.8
	20	1.5	1.8	1.7		3.8
		5	20	1.9		3.8
		10	80	2.0		3.8
		Intermediate-Therapy and Deep-Therapy Units				
h.v.l. mm.	TSD cm.		Area cm.²	D1/2 cm.	D9/10 cm.	Fall-off Ratio
1.0 mm. Al	15		5	1.0	4.8	4.8
			25	1.3	5.5	4.2
			100	1.5	6.3	4.2
			200	1.6	6.5	4.1
	20		5	1.0	5.0	5.0
			25	1.5	6.0	4.0
			100	1.7	6.7	3.9
			300	1.8	7.5	4.2
	30		5	1.1	5.3	4.8
			25	1.5	6.3	4.2
			100	1.8	7.5	4.2
			300	1.9	8.3	4.4
2.0 mm. Al	15		5	1.4	6.0	4.3
			25	1.9	7.0	3.7
			100	2.2	8.0	3.6
			200	2.4	8.3	3.5
	20		5	1.6	6.5	4.0
			25	2.1	7.3	3.5
			100	2.5	8.7	3.5
			300	2.7	9.3	3.4
	30		5	1.7	7.0	4.1
			25	2.4	8.0	3.3
			100	2.8	9.5	3.4
			300	3.1	10*	3.2*
3.0 mm. Al	15		5	1.7	6.7	3.9
			25	2.4	7.7	3.2
			100	2.7	9.0	3.3
			200	2.9	9.5	3.3
	20		5	1.9	7.0	3.7
			25	2.6	8.0	3.1
			100	3.1	9.7	3.1
			300	3.3	11*	3.3*
	30		5	2.0	7.7	3.9
			25	2.9	9.0	3.1
			100	3.5	11*	3.1*
			300	3.7	12*	3.2*
4.0 mm Al	15		5	2.0	7.0	3.5
			25	2.7	8.3	3.1
			100	3.2	9.5	3.0
			200	3.3	10	3.0
	20		5	2.1	7.7	3.7
			25	3.0	9.0	3.0
			100	3.6	10*	2.8*
			300	3.8	11*	2.9*

Table continued on page 82

TABLE I: D1/2 AND FALL-OFF RATIO AS FUNCTIONS OF H.V.L., T.S.D., AND PORTAL AREA.—*Cont.*

h.v.l. mm.	T.S.D. cm.	Area cm.²	D1/2 cm.	D9/10 cm.	Fall-Off Ratio
		Intermediate-Therapy and Deep-Therapy Units			
4.0 mm. Al *Cont.*	30	5	2.4	8.5	3.5
		25	3.5	10	2.9
		100	4.1	12*	2.9*
		300	4.4	13*	3.0*
6.0 mm. Al	15	5	2.1	8.0	3.8
		25	3.0	9.0	3.0
		100	3.7	11*	3.0*
		200	3.8	11*	2.9*
	20	5	2.5	8.7	3.5
		25	3.4	10	2.9
		100	4.2	12*	2.9*
		300	4.5	13*	2.9*
	30	5	2.7	9.5	3.5
		25	3.9	11*	2.8*
		100	4.8	13*	2.7*
		300	5.3	15*	2.8*
	40	5	2.9	10	3.5
		25	4.0	12*	3.0*
		100	5.2	14*	2.7*
		300	5.7	16*	2.8*
8.0 mm. Al or 0.44 mm. Cu	15	5	2.4	8.5	3.5
		25	3.3	9.7	2.9
		100	4.2	12*	2.9*
		200	4.4	12*	2.7*
	20	5	2.7	9.3	3.4
		25	3.7	11*	3.0*
		100	4.7	13*	2.8*
		300	5.1	14*	2.7*
	30	5	3.1	10	3.2
		25	4.3	12*	2.8*
		100	5.4	14*	2.6*
		300	6.1	17*	2.8*
	50	5	3.5	11*	3.1*
		25	4.7	13*	2.8*
		100	6.1	15*	2.5*
		300	6.9	18*	2.6*
1.0 mm. Cu	15	10	3.6	12	3.3
		25	4.1	13	3.2
		100	5.2	15	2.9
		400	6.3	18	2.9
	25	10	3.9	12	3.1
		25	4.6	14	3.0
		100	5.8	16	2.8
		400	7.0	19	2.7
	50	10	4.3	13	3.0
		25	5.0	15	3.0
		100	6.4	17	2.7
		400	8.0	20	2.5
2.0 mm. Cu	15	10	3.6	12	3.3
		25	4.3	14	3.3
		100	5.3	17	3.2
		400	6.6	21	3.2
	25	10	3.9	13	3.3
		25	4.8	15	3.1
		100	6.1	18	3.0
		400	7.6	22	2.9
	50	10	4.3	14	3.3
		25	5.3	16	3.0
		100	6.9	19	2.8
		400	8.7	23*	2.6*

TABLE I: D1/2 AND FALL-OFF RATIO AS FUNCTIONS OF H.V.L., T.S.D., AND PORTAL AREA.—*Cont.*

h.v.l. mm.	T.S.D. cm.	Area cm.[2]	D1/2 cm.	D9/10 cm.	Fall-Off Ratio
		Intermediate-Therapy and Deep-Therapy Units			
2.0 mm. Cu. *Cont.*	70	10	4.6	15	3.3
		25	5.6	17	3.0
		100	7.4	20	2.7
		400	9.6	25*	2.6*
4.0 mm. Cu	50	10	5.0	16	3.2
		25	6.0	17	2.8
		100	7.4	20	2.7
		400	9.0	25*	2.8*
	70	10	5.4	17	3.2
		25	6.5	19	2.9
		100	8.2	22*	2.7*
		400	10	26*	2.6*
		Megavoltage Therapy Units			
8.0 mm. Cu	100	20	8.0		
		50	8.8		
		100	9.8		
		400	11		
10.0 mm. Cu	100	20	8.9		
		50	9.9		
		100	11		
		400	12		
12.0 mm. Cu	100	20	9.9		
		50	11		
		100	12		
		400	13		
15.0 mm. Cu	100	20	11		
		50	12		
		100	13		
		400	14		

* Extrapolated data.

due, in part, to the fact that different workers have employed different phantom materials (wax, "Mix D," polystyrene, water, etc.) and in part to the fact that different measuring devices have been used (nylon Victoreen chambers, Kustner chambers, and mesh-type chambers). Moreover, most of the reported studies cover a very limited range of portal areas and target-skin distances.

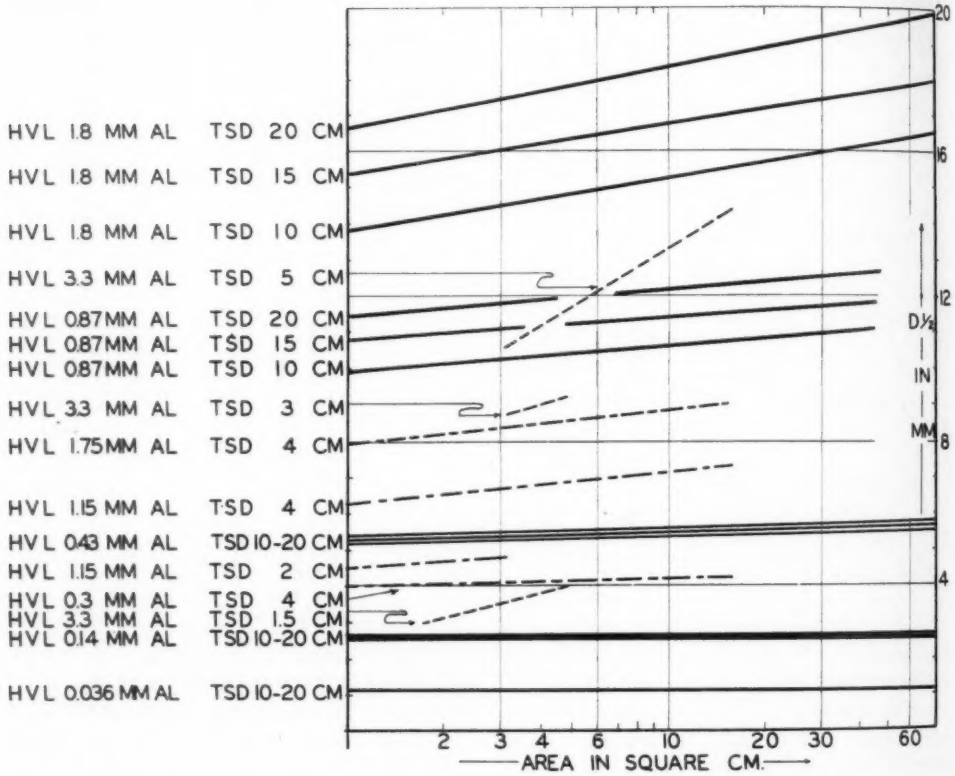
The most comprehensive and carefully evaluated data pertaining to Chaoul and Philips units are those collected by workers at the Siemens-Reiniger-Werke and by W. J. Oosterkamp, respectively, published in the *British Journal of Radiology*, Supplement 5 (3). The values of D1/2 for such units presented here are based on these measurements. Inconsistency in slopes of the curves of Figure 1 probably reflects variations in the technic of dosimetry by the various investigators.

Jennings' data (5, 6) relative to beryllium-window tubes appear to be the most definitive measurements available, and have been taken as the basis for the values given here for the beryllium-window unit.

Values for D1/2 in the supervoltage range are also based on data from the *British Journal of Radiology*, Supplement 5, because again these data appear to be more comprehensive and more carefully evaluated than any other available measurements.

3. Calculations: The values of D1/2 were obtained, in general, by arithmetic interpolation of the depth-dose data cited above. However, so far as is known, no published data exist for half-value layers of 1 and 2 mm. Cu at target-skin distances of less than 40 cm., nor are data known to be available for radiation having a half-value layer of 0.44 mm. Cu at a target-skin distance of 50 cm. Values of D1/2 corre-

$D\frac{1}{2}$ AS A FUNCTION OF HALF VALUE LAYER TARGET SKIN DISTANCE AND PORTAL AREA IN SUPERFICIAL THERAPY.



FACTORS YIELDING SPECIFIED HALF VALUE LAYERS:

HVL SPECIFIED (MM. AL)	0.036	0.14	0.3	0.43	0.87	1.15	1.75	1.8	33
GENERATOR	BERYLLIUM WINDOW	BERYLLIUM WINDOW	PHILIPS	BERYLLIUM WINDOW	BERYLLIUM WINDOW	PHILIPS	PHILIPS	BERYLLIUM WINDOW	CHAOUL
KILOVOLTS	50	50	44	50	50	44	44	50	60
ADDED FILTER (MM. AL)	0.0	0.15	0.0	0.4	1.0	1.0	2.5	3.0	0.0

Figure 1

(Subsequent to the preparation of this chart, the h.v.l. given as 0.036 mm. Al was redetermined to be 0.07 mm. Al. The revised value has been used in Table I.)

sponding to these distances were, therefore, calculated from available depth-dose data by the method of Mayneord and Lamerton (9). Also, because of the extremely rapid fall-off of the tissue dose with depth in the cases of the contact therapy beams, arithmetic interpolation seemed inadequate. Accordingly, for the Philips, Chaoul, and beryllium-window units, the basic depth-dose data were plotted and

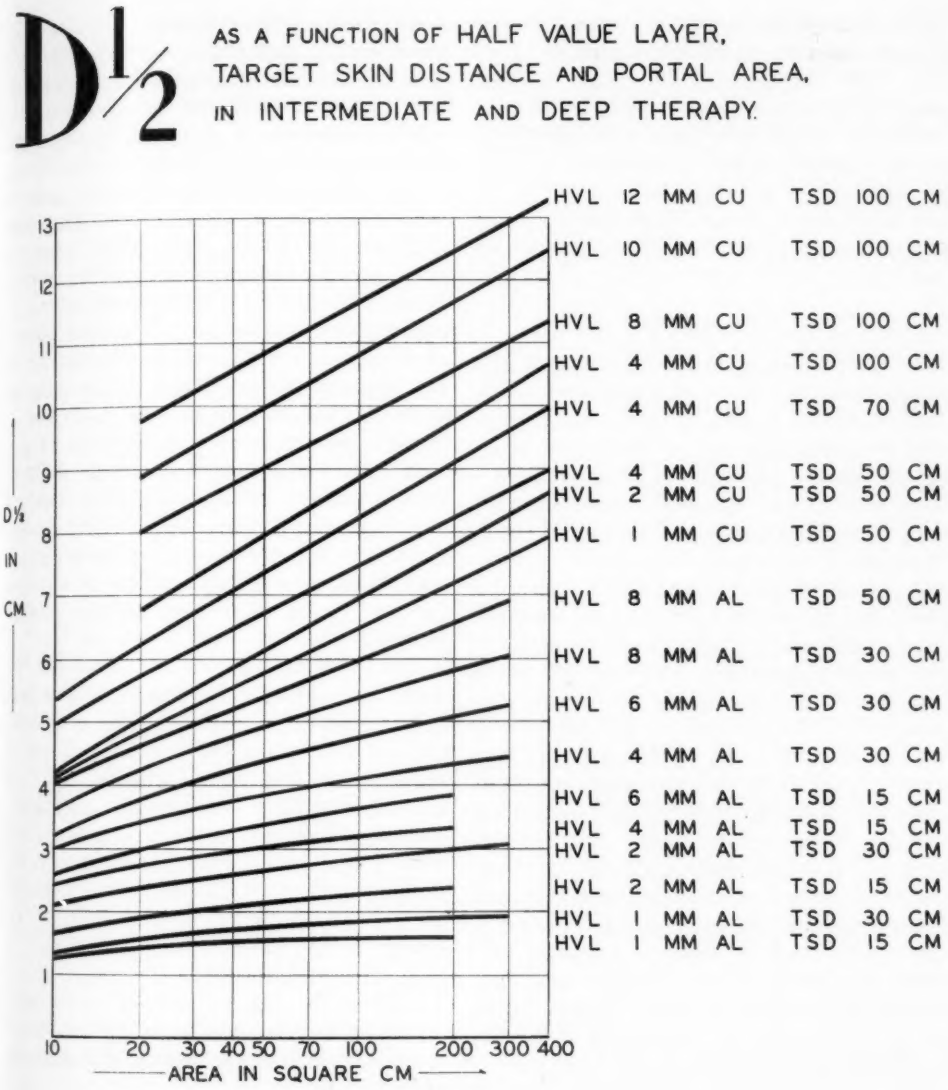


Figure 2

D1/2's (corresponding to the appropriate half-value layers, portal areas, and target-skin distances) were determined graphically.

Values for D9/10 were obtained similarly except that in many cases it was necessary to extrapolate the published data graphically. Data procured by extrapolation are so indicated in Table I. Fall-off ratios were calculated from the resulting values for D9/10 and D1/2 except in the case of the beryllium-window unit, for which Jennings' previously published values were employed. D9/10's and fall-off ratios were not calculated for supervoltage beams, since the evaluation of D9/10 would require extrapolation over several centimeters, making the data of doubtful significance.

All of the data in Table I are expressed in terms of two significant figures.

4. The Graphic Data: The accompanying graphs (Figs. 1 and 2) are intended as visual aids for use in radiation-therapy planning and in teaching. They present values of D1/2 as a function of half-value layer, target-skin distance, and portal area, and are based on the data in Table I.

DISCUSSION

1. In planning superficial therapy, the objective is to deliver as much as possible of the ionizing radiation to the zone occupied by the lesion, and as little as possible to the deeper structures. Since the attenuation of the incident radiation with depth is approximately exponential, it is impossible to deliver an adequate dose to the base of a lesion and still achieve the ideal condition in which no radiation reaches the underlying structures. Hence, one must compromise between adequate dosage to the base of the lesion and the possibility of damage to deeper structures. According to Jennings (5, 6) the most satisfactory compromise in most cases is to deliver 50 per cent of the dose on the skin to the lesion's base. It is for this reason that D1/2, which takes into account the clinical factors of target-skin distance and portal area, is a useful way to specify the quality of a beam. Theoretically then, according to this author, radiation should be selected of such a quality that its D1/2 equals the estimated depth of the lesion to be treated.

Since the radiation penetrating beyond the half-value depth is unwanted and, indeed, damaging to healthy tissue, Jennings argues that the more rapidly this remnant radiation is attenuated, the less the harm done to deeper, healthy structures. An expression of the rate of absorption of this remnant radiation, as a function of depth in tissue, is obtained by dividing the tissue depth corresponding to 90 per cent absorption (D9/10) by that corresponding to 50 per cent absorption (D1/2), and it is this ratio that Jennings has defined as the fall-off ratio. For radiation of any given D1/2, it may be said that, if the fall-off ratio is small, the distribution of radiation in the tissues is relatively favorable; if the ratio is large, the dose distribution is relatively unfavorable.

The data presented, therefore, are of practical value in planning the treatment of superficial lesions, since, when the depth of a lesion is estimated, and the portal area to be treated is determined, the physical factors (half-value layer and target-skin distance) which will yield radiation of the appropriate D1/2 may be ascertained with the aid of the graphs in Figure 1. Where two or more combinations of factors yield radiation of the desired D1/2, it is possible to determine from Table I whether or not there is a significant difference in the corresponding fall-off ratios and, if so, which combination of physical factors yields the most favorable dose distribution.

For example, it is apparent from the data that only the beryllium-window unit without added filtration produces an optimum D1/2 (1 mm.) for the treatment of corneas to prevent vascularization. On the other hand, a skin lesion 1 to 1.5 cm. deep and 6 sq. cm. in area, may be treated with the beryllium-window unit, the Chaoul unit, or a 135 kilovolt intermediate-therapy unit if the appropriate external filtration is selected. In such a case, the selection of a treatment plan is governed by the radiologist's experience with the various units, their comparative convenience, and the relative fall-off ratios of the beams.

2. The application of the data to the calculation of integral dose is straightforward. Mayneord (10) defines the integral dose as the total energy absorbed by the body throughout the entire irradiated volume and shows that this is approximately equal, for total absorption of the radiation beam, to the product of the surface dose (D_0), the portal area (A), the D1/2, and a constant, 1.44. Thus, to a first approximation, we have the formula:

$$I = 1.44 \times D_0 A\ (D1/2).$$

It must be emphasized that this equation neglects the divergence of the beam and assumes flat isodose curves and total absorption of the incident radiation within the irradiated tissue. For a detailed discussion of the derivation of the equation, its limitations, and the correction factors to be applied for more precise calculation of integral dose, the reader is referred to Mayneord's original publication.

3. To the author's knowledge, fall-off ratios have not previously been tabulated for half-value layers above 2 mm. of Al, though the variation in fall-off ratio with portal area and target-skin distance at higher half-value layers is of some interest. It is to be noted that the fall-off ratio is smaller (more favorable) for high half-value layers than for less penetrating radiation.

SUMMARY

1. Values of half-value depth and fall-off ratio are presented for a wide range of half-value layers, portal areas, and target-skin distances.

2. The usefulness of the data in teaching, in treatment planning, and in calculating integral dose is discussed.

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SUMMARIO IN INTERLINGUA

Profundor de Medie Valor e Ratio de Declino Como Functiones de Area Portal, Distantia de Scopo a Pelle, e Spissitate de Medie Valor

Per medio de un longe tabula e 2 graphicos, iste articulo presenta in forma commode valores de profundor de medie valor e de ratio de declino correspondente al integre scala de qualitates radiational, areas portal, e distantias de scopo a pelle que occurre con ulle grado de probabilitate in roentgenotherapia clinic.

Iste datos se ha provate utile in specificar fasces de radiation superficial in le planation de cursos de tractamento, in provider un indice del distribution de radiation in le tessutos in therapia profunde e intermediari, e in facilitar pro le studente le visualisation del effecto de alterationes de kilovoltage, de filtro externe, de dimension portal, e de distantia de scopo a pelle super le distribution del radiation in le tessutos. Finalmente, iste datos es de adjuta in le computation del dose integral.

A Simplified Ruler Method for Dosage Calculation in Rotational Therapy in the Intermediate Voltage Range[1]

WILLIAM A. O'SHEA, Jr.,[2] M.D., C. H. CHANG, M.D., and FRANKLIN HUTCHINSON, Ph.D.

THE PURPOSE OF this paper is to describe a ruler which will simplify dosage calculations in intermediate-voltage rotation therapy. One ruler supplies the tumor and skin dose information for a given combination of physical factors (half-value layer, target-axis distance, and field area). The use of similar rulers for central dose determination in supervoltage rotation has been described by Braestrup and Mooney (1), but no such ruler is available in the intermediate voltage range.

The advantages of the ruler method over others (2–6) which supply skin and tumor dose information is that the ruler is simple to make and easy to use. Tumor and skin dose calculations are usually accomplished in a few minutes.

Integral dose calculations and off-center dose determinations require information not obtained with this ruler. More complicated technics and a large number of isodose charts are needed for that purpose.

With shortening of the time required to set up and calculate the essential dosage information, it is hoped that rotation therapy will be more widely used in general hospitals.

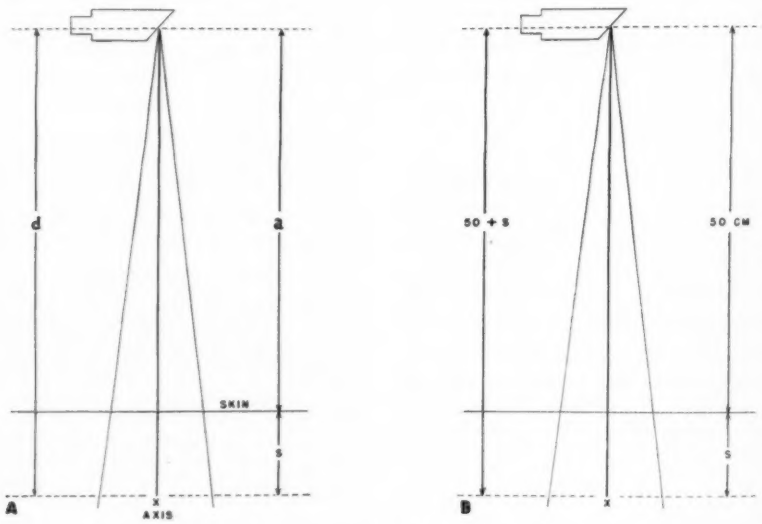

Fig. 1. Schematic drawing showing relation of air, skin, and tumor doses to dosage calculation in rotational therapy.

METHOD OF DOSAGE CALCULATION AND RULER CONSTRUCTION

The dose received by a tumor s centimeters under the skin and d centimeters from the target (Fig. 1A) can be calculated under the following assumption used by Kligerman, Rosen and Quimby (2). The dose D_d at the axis as shown is

$$D_d = D_a \frac{a^2}{d^2} f_A(s) \qquad (1)$$

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TABLE I: CALCULATED PERCENTAGE TUMOR AND SKIN DOSES AT VARIOUS TISSUE DEPTHS
(5 × 12 cm. port size at 50 cm. T.S.D.; 1.5 mm. Cu h.v.l.; 70 cm. target-axis distance)

Tissue Depth (cm.)	Corrected Port Size on Skin (A cm.^{2})	Per Cent Depth Dose (D_s) at 50 cm. T.S.D. (from Standard Table)	Tumor Dose (TE) Factor $\frac{(50+s)^2}{70^2}$	Per Cent Tumor Dose (TD)	Per Cent Skin Dose (D_0) at 50 cm. T.S.D. (from standard Table)	Skin Dose (SD) Factor $\frac{50^2}{(70-s)^2}$	Per Cent Skin Dose (SD)
1	114	130	53	69	132	53	70
2	111	119	55	65	132	54	71
3	108	106	57	61	131	56	73
4	105	94	60	56	131	57	75
5	101	82	62	51	131	59	77
6	98	72	64	46	131	61	80
7	95	62	66	41	131	63	83
8	92	55	69	38	130	65	85
9	89	47	71	33	130	67	88
10	87	40	74	30	130	70	91
11	84	35	76	27	130	72	94
12	81	30	79	24	129	75	97
13	78	26	81	21	129	77	100
14	75	22	84	18	129	80	103
15	73	19	86	16	129	83	107
16	70	16	89	14	128	86	110
17	67.5	13	92	12	128	89	114
18	65	11	95	11	128	93	119
19	62.5	10	97	10	128	97	124
20	60	9	100	9	127	100	127

where D_a is the air dose at any convenient reference distance, a cm., from the target. $f_A(s)$ is the fraction by which the dose is reduced by passing through s cm. tissue. The subscript A is appended to indicate that this fraction depends on the area of the field on the skin. It is assumed that $f_A(s)$ does not depend on the target-skin distance.

To evaluate $f_A(s)$ from the standard central axis depth dose data (5) for depth dose $D_{(50+s)}$ at 50 cm. target skin distance (Fig. 1B):

$$D_{(50+s)} = D_{50}\frac{50^2}{(50+s)^2}f_A(s) \qquad (2)$$

or

$$f_A(s) = \frac{D_{(50+s)}}{D_{50}}\frac{(50+s)^2}{50^2} \qquad (3)$$

Since $f_A(s)$ does not depend on distance d, we can now calculate the dose at any target-tumor distance in terms of the dose in air at 50 cm. (or any convenient distance, a cm., we prefer), using (1) and (3)

$$D_d = D_{50}\frac{50^2}{d^2}\frac{D_{(50+s)}}{D_{50}}\frac{(50+s)^2}{50^2} = D_{(50+s)}\frac{(50+s)^2}{d^2} \qquad (4)$$

The tumor dose at s cm. below the skin in a target-axis distance of, say, 70 cm. will then be obtained by:

1. Calculating average field area, A, of the beam on the skin.

2. Looking in the standard depth dose tables for $D_{(50+s)}$, *i.e.*, the dose at s cm. below the skin for a field area of A cm^2 and target-skin distance of 50 cm. Since our x-ray machines are calibrated at 50 cm., and since the ports used are designated by their size at 50 cm., we have adopted the convention of expressing both tumor and skin doses in percentage of the air dose at 50 cm. from the target.

3. Multiplying $D_{(50+s)}$ by $\frac{(50+s)^2}{70^2}$

The calculated dose is then the dose delivered at s cm. below the skin in a target axis distance of 70 cm. per 100 r in air at 50 cm. target-skin distance.

The skin dose may be obtained by multiplying the dose obtained from the standard table by $\frac{50^2}{(70-s)^2}$, inasmuch as the skin dose for any port size does not change with the target-skin distance. The correction should be applied for increasing area

covered by the beam as the skin gets farther away from the end of the treatment cone at 50 cm. The calculated percentage tumor dose and skin dose at various depths, with corresponding corrected portal areas on the skin, for one given combination of physical factors, are presented in Table I. The percentage skin dose and tumor dose data are inscribed on the ruler to be described below.

The ruler is made of transparent plastic and is inscribed with two scales, one for the percentage skin doses and the other for the percentage tumor doses at the various tissue depths, expressed in cm. When the ruler is placed on a full-scale cross-section contour tracing of the patient (Fig. 2) with its zero point at the center of the tumor and the central line along the beam axis, the intercept of one scale with the skin surface gives the skin dose, the other the tumor dose. One ruler supplies such information only for one given combination of physical factors. Therefore, several sets of rulers for different field size, half-value layer, and target-axis distance are usually made for the common clinical conditions encountered.

PROCEDURE AND EXAMPLE OF DOSAGE APPROXIMATION

An esophageal tumor is located fluoroscopically with whatever contrast material is necessary at four points on the body surface at the level of the center of the tumor, the four points being the extremities of two lines through the tumor at right angles to each other.

A plaster strip is wrapped around the patient at this level and the four points are marked on the plaster. When the plaster has hardened, it is cut at one point and removed. A contour tracing is made of its inner surface on a piece of transparent paper and the four points are marked on the edge of the tracing. Connection of the opposing points will then locate the center of the tumor in the tracing (Fig. 2).

By putting a pin through the zero point of the ruler into the center of the tumor, as shown on the tracing, and rotating the ruler, the skin and tumor dose readings can be obtained from the points where the contour tracing intersects the scales of the ruler.

The number of measurements taken is generally considered to be one for every 30°. A division of the circle into twelve segments was found to be adequate in most cases; the greater the number of divisions, the greater will be the accuracy of the final average.

For estimation of tumor dose, the average tumor dose from all segments is computed. For skin dose the average skin dose from all segments is again divided by the number of segments taken, since one segment on the skin is in the beam one-twelfth of the time.

Segment	TD	SD
1	31	90
2	26	95
3	17	105
4	16	107
5	20	101
6	22	99
7	29	92
8	26	95
9	22	99
10	17	106
11	19	102
12	25	95
	270	1186

Average tumor dose: $\frac{270}{12} = 22.3$ r per 100 r in air at 50 cm.

Average skin dose $\frac{1186}{12 \times 12} = \frac{1186}{144} = 8.24$ r per 100 r in air at 50 cm.

To calculate the air dose at 50 cm. required to give a daily tumor dose of 150 r:

$$\frac{\text{required air dose at 50 cm.}}{150 \text{ r}} = \frac{100}{\text{calculated TD/100 r in air at 50 cm.}}$$

$$\frac{x}{150} = \frac{100}{22.3}$$

$$x = 673 \text{ r}$$

If the dose rate in air at 50 cm. is 103 r/min, each treatment will last $\frac{673}{103} = 6.53$ min.

This will give an average skin dose of $6.53 \times 8.24 = 55$ r per treatment day.

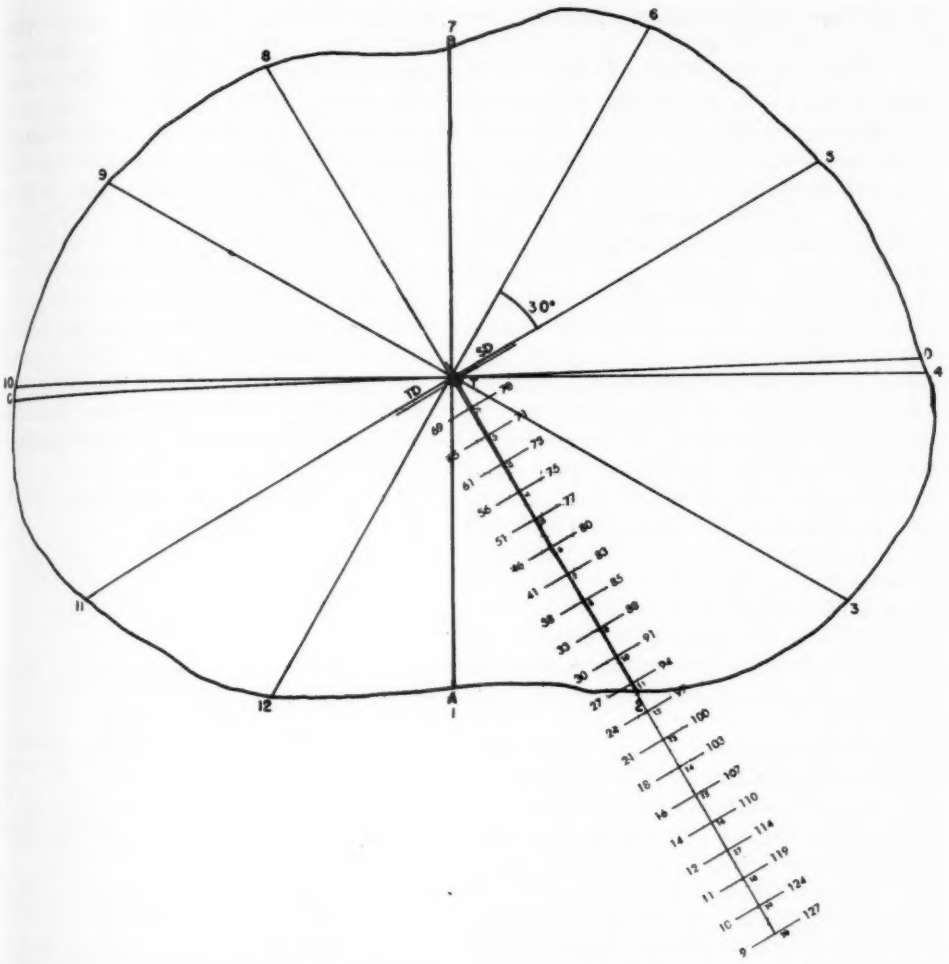

Fig. 2. Application of ruler to a full-scale cross-section contour tracing of the patient for determination of tumor and skin doses. Physical factors pertaining to this ruler: 5 x 12 cm. port.; 1.5 mm. Cu. h.v.l.; 70 cm. T.S.D. Doses as per cent of r in air at 50 cm.

DISCUSSION

Rotational therapy in the 250-kv range has in the past required time-consuming dose calculations. The ruler method described here, similar to that used by Braestrup in rotating telecobalt equipment, greatly simplifies the central tissue dose determination and provides an accuracy adequate for clinical purposes in a general hospital where the help of a radiation physicist is not always available.

The percentage tissue dose and skin dose along the axis of rotation can be easily calculated from the standard central axis depth dose tables, assuming that the attenuation factor f_A (s) does not depend on the target-skin distance. For a rotational target axis distance of 70 cm., the tissue dose can be expressed in r per 100 r in air at 50 cm. target skin distance by multiplying the depth value obtained from the standard table at depth s by $\frac{(50 + s)^2}{70^2}$; the skin dose by multiplying

the skin dose value obtained from the standard tables by $\frac{50^2}{(70 - s)^2}$. Rulers based on these data give directly the center dose and skin dose for different tissue thicknesses.

In rotational therapy, the radius of rotation (target-axis distance) is usually constant, so that target-skin distance (T.S.D.) varies with the angular position of the beam, depending on the outline of the patient. As the T.S.D. varies, so does the surface area of the field. Since the attenuation factor, f_A (s), varies with the area, A, of the field on the skin, a correction should be applied for the increasing area covered by the beam as the skin moves further away from the end of the treatment cone, as well as for the variation produced by the angular position of the beam during rotation. The field area correction is a prerequisite for the use of the standard depth dose tables. An average field area obtained by averaging one maximum and one minimum value from the 12 segments is reasonably accurate and acceptable.

Recently, Wheatley (6) has described a simple numerical system of dosage estimation based on central axis depth dose data. While it is less time-consuming than other methods involving only numerical manipulation, an estimation of dose at three or four points of interest still requires more than half an hour according to the author.

Castro, Soifer and Quimby's approximation method (3) with the use of isodose charts offers a reasonably simple and quick means of dose estimation at any point of interest in the rotational field. Their method should be employed when information for doses other than the central tumor dose and skin dose is desired. In general, however, for a centrally located tumor, dosage calculation according to the ruler method described in this paper will be found adequate.

SUMMARY

A simple method of dosage calculation based on the standard central depth dose data has been described. Such calculations have been used for construction of a ruler to give central tumor dose and skin dose values for vertical rotation therapy in the intermediate voltage range.

An example of the application of the ruler method is given.

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SUMMARIO IN INTERLINGUA

Un Simplificate Methodo a Regula-Norma pro le Calculation de Dosages in Therapia Rotational a Voltages de Ordine Intermediari

Ha essite elaborate un methodo a regula-norma pro le calculation del dose cutanee e del dose tumoral in therapia rotational a nivellos del ordine de 250 kv. Le calculationes del dosage es facite per medio de tabulas standard de dosage de profunditate a axe central e es inscribite in un regula-norma de plastico. Le dosages es exprimite in pro cento de dose aeree a 50 cm ab le scopo.

Es presentate un exemplo del application del methodo al tractamento de un tumor

esophagee. Le tumor es locate fluoroscopicamente, e 4 punctos es marcate al superficie del corpore, correspondente al extremitates de duo lineas que passa a transverso le tumor a angulos recte le un al altere. Un banda de emplastro es volvite circa le patiente al nivello del tumor, e le quatro punctos es marcate in le emplastro. Per medio de isto, un traciage de contorno de dimensiones natural es facite, e in illo le position del tumor es indicate. Un agulia es plantate a transverso le puncto de zero del regula-norma a in le centro del tumor assi indicate. Le regula-norma es rotate, e le doses cutanee e le doses tumoral es determinate ab le punctos de intersection del traciage con le scala del regula-norma. Estimationes es facite pro cata un de un dozena de segmentos del contorno. Lor magnitude medie es usate como dose tumoral. Pro obtener le dose cutanee, le magnitude medie es dividite de novo per le numero del segmentos, proque cata un del segmentos cutanee se trova sub le fasce de radiation solmente un dece-secunde parte del tempore.

Per medio de iste methodo, le calculation del doses tumoral e cutanee es usualmente effectuabile intra alicun minutas. Le methodo non es applicabile a calculationes de doses integral e a calculationes excentric.

Excretion Cholecystocholangiography

A Study Based on 1,400 Cases Examined with Telepaque and/or Cholegrafin[1]

GONZALO ESGUERRA-GÓMEZ, M.D.

THE FREQUENT demonstration of the biliary ducts following the advent of Telepaque (1) and the magnificent visualization obtained with Cholegrafin (2) gave rise to a large series of scientific communications (3) indicating the importance of these examinations and leading consequently to their routine employment by radiologists everywhere.

Although many writers speak of oral or intravenous cholangiography (1–16), the term cholecystocholangiography (17, 18) is more appropriate. To differentiate the procedure from the cholangiography done at operation or with the aid of laparoscopy, value of the normal radiologic examination of the biliary passages from the clinical point of view.

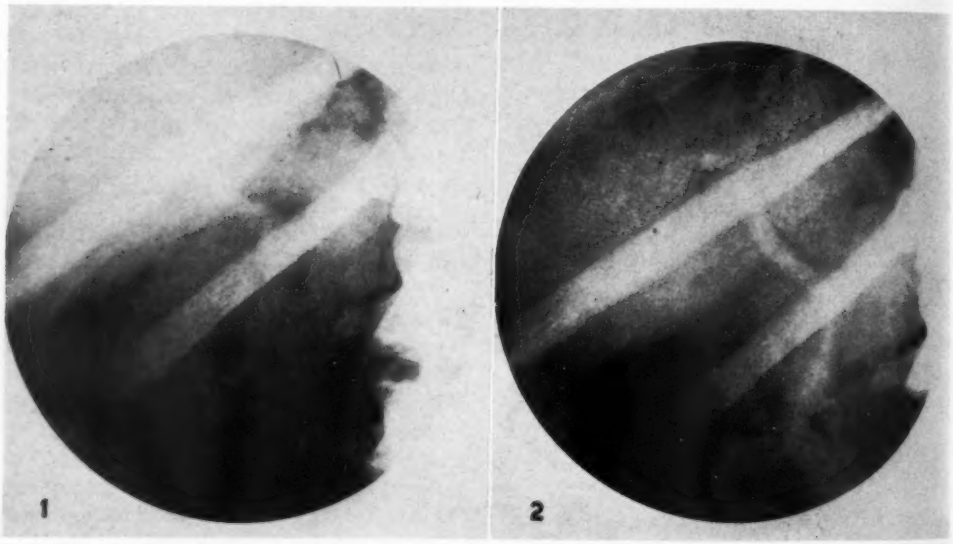


Fig. 1. Cholecystocholangiogram obtained with Telepaque (6 gm.), showing normal ducts following cholecystectomy.

Fig. 2. The same case as in Fig. 1, examined with Cholegrafin. The duct shadow is much more "contrasty."

The present work proposes to set forth the results obtained in a group of 1,400 private patients from the civil population, in whom oral procedure was combined with the intravenous method to determine the normals which in our opinion should be sought routinely in such studies; to learn to recognize the shadows which in certain cases of congenital anomalies serve the surgeon as a guide to successful operative intervention; and to discuss the it has seemed to us that we should call it "excretion cholecystocholangiography." This single term includes examination both of the gallbladder and the ducts. In our opinion, in spite of the fact that the substances employed and the methods of introduction into the organs are different, we are dealing with a single radiologic study, which can be accomplished by either means alone or with both at the same time.

[1] From Clínica de Marly, Bogotá, Colombia. Read before the Eighth International Congress of Radiology, Mexico City, July 1956. Accepted for publication in December 1956.

TABLE I: TYPE OF EXAMINATION IN 1,400 CASES

Telepaque	1,218(87%)
Telepaque and Cholegrafin	114(8.14%)
Cholegrafin alone	68(4.86%)
Examined with Telepaque	1,332(87.98%)
Examined with Cholegrafin	182(12.02%)

TABLE II: GALLBLADDER VISUALIZATION IN 1,332 CASES EXAMINED WITH TELEPAQUE

Telepaque	1,332 cases
Gallbladder visible	1,202(90.24%)
No gallbladder shadow (gallbladder excluded)	130(9.76%)
Good gallbladder visualization	1,125(84.45%)
Deficient gallbladder visualization	77(5.79%)
No gallbladder shadow	130(9.76%)

TABLE III: VISUALIZATION OF THE DUCTS IN 1,202 CASES IN WHICH THE GALLBLADDER WAS VISUALIZED BY TELEPAQUE

Visualization of the ducts	995(82.78%)
Visualization of the trunk of the hepatic duct	199(20%)*
Failure of canalicular visualization	207(17.22%)

* In our opinion, visualization of the hepatic trunk without pathologic dilatation of the common duct may be considered as normal (Fig. 3).

TABLE IV: SUMMARY OF FINDINGS IN 1,332 CASES EXAMINED WITH TELEPAQUE

Gallbladder excluded (no gallbladder shadow)	130(9.76%; with ducts visible 4, or 3.08%)
Gallbladder visualized	1,202(90.24%)
Normal	833(69.30%)
Pathologic	369(30.70%)
Stones in gallbladder	221(59.87%)
Ductal stones	3(0.82%)
Atonic or hyperkinetic gallbladders	92(24.94%)
Other pathologic states (odditis, papillomas, cholesterosis, etc.)	53(14.37%)

TABLE V: CONGENITAL ANOMALIES OF THE BILIARY TRACT DEMONSTRATED BY TELEPAQUE

Opening of the cystic duct on the left border of the hepatic duct	19(1.9%)
Congenital gallbladder adhesions	20
Bilobar or biloculate gallbladders	3
Divided gallbladder	1
Septal gallbladder (Hill)	1
Volvulus of the gallbladder	1
Accessory hepatic duct	1
Diverticula of the gallbladder	3 (Figs. 9 and 10)
Diverticula of the common duct	1 (Fig. 11)
Diverticula of the cystic duct	1 (Fig. 12)

We begin our examination with the oral method, using Telepaque, (*a*) because ingestion of the contrast material the preceding afternoon is more easily accomplished than intravenous injection in the office; (*b*) because failure of visualization of the gallbladder when this organ is permeable shows that its functions of concentration have been lost; (*c*) because the degree of density is an index of the manner in which these functions have been carried out (20).

TABLE VI: EXCRETION CHOLECYSTOCHOLANGIOGRAPHY WITH CHOLEGRAFIN

Cholecystectomized patients	
Without shadows of biliary elimination	10(15.38%)
Ductal visualization	55(84.62%)
Normal	49%
Pathologic	51% (gallstones, 46.43%)
Non-operative cases	
Without shadows of biliary elimination	17(14.53%)
With excretion shadows	100(85.47%)
Normal	13.67%
Pathologic	71.80% (gallstones, 57.14%)

When the oral procedure fails to produce a gallbladder shadow during elimination of the medium, recourse is had immediately to Cholegrafin, since a large number of gallbladders not shown by Telepaque fill very well with the intravenous preparation (12), and because visibility of the ducts is good in the presence of a vesicular block or when the patient has been cholecystectomized. In such cases we advise that the examination be completed in this one session.

In cholecystectomized patients we have often used Telepaque, because, with the technic advised by Twiss and his collaborators (22), and very often with routine examinations, shadows of the common duct and the hepatic trunk duct may be obtained. Not only is this true of patients who have been cholecystectomized but also of those whose gallbladders have been non-functioning for some time. However, since these canalicular structures are visualized to better effect in this group (Figs. 1 and 2) with Cholegrafin, we do not believe that an initial oral study is indispensable.

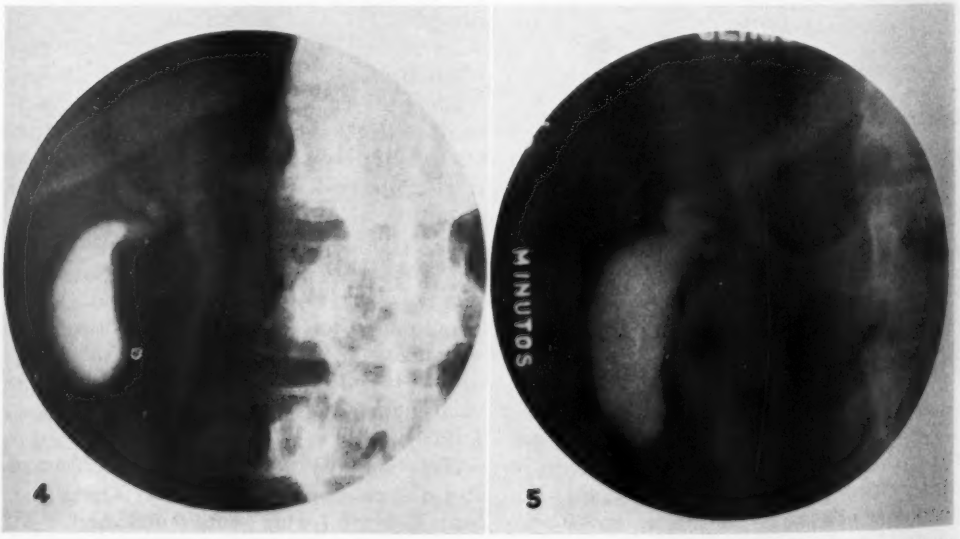
Tables I to V show the results obtained in 1,332 patients examined with Telepaque, and Table VI the results obtained with Cholegrafin.

Among the congenital anomalies, special

Fig. 3. Telepaque. Gallbladder and ducts normal. Injection of the trunk of the hepatic duct. The cystic duct empties on the anterior aspect of the hepatic duct.

attention is called to the occurrence of the outlet of the cystic duct on the left border of the hepatic duct after bordering the posterior aspect of the latter (Figs. 4 and 5). The diagnosis of this anomaly is of the greatest importance, since it is often overlooked at the time of operation by reason of membranes surrounding the termination of the cystic duct and the corresponding zone of the hepatic. For this reason, the surgeon often leaves a fragment of the cystic duct, which can give rise to the reconstruction of a rudimentary gallbladder, an occurrence which has been proved in 4 of our cholecystectomized patients.

According to the anatomic studies of Descomps (24), this anomaly is found in 8 per cent of cases, but we were able to diagnose it in only 1.9 per cent of our series. This is due to the fact that the diagnosis can be made only when the trunk of the hepatic duct has been visualized (Fig. 6); even then, on occasion, superimposition of the inferior portion of the trunk of the hepatic and the outlet of the cystic duct is so exact that it is not possible to make a diagnosis. When this anomaly is suspected, therefore, it is necessary to obtain roentgenograms in various angles of obliquity. We have reached the conclusion that when the cystic duct is too long (Figs. 7 and 8), it almost always coincides with an abnormal outlet on the common duct. We believe that this diagnosis will be made more frequently if one



Figs. 4 and 5. Telepaque. Gallbladder and ducts normal. The cystic duct in each instance empties on the left border of the hepatic duct.

TABLE VII: RESULTS OBTAINED WITH TELEPAQUE AND/OR CHOLEGRAFIN

Total of cases examined with Telepaque and/or Cholegrafin	1,400
Diagnosis made in	1,373(98.07%)
No biliary excretion shadows obtained	27(1.93%)
Normal cases	908(64.86%)
Pathologic cases	492(35.14%)

simply keeps in mind that it may be coincident with a very long cystic duct and continues with the examination until the trunk of the hepatic is visualized. Since the hepatic duct alone is visible in 20 per cent of persons examined, the least percentage encountered will be proportional to this visibility. A résumé of the congenital anomalies in the present series is presented in Table V.

For the purpose of evaluating results obtained with Cholegrafin exclusively and those with a combination of Cholegrafin and Telepaque, we have compared our findings with the two preparations with those of Hornykiewytsch and Stender (14) in 1,000 patients examined only with Cholegrafin (Table VIII). Also it seemed of interest to compare 500 cases studied with Telepaque by Whitehouse and Martin (26) with the 1,332 cases examined by us with the same medium, for the results are very similar.

Fig. 6. Telepaque. Gallbladder and ducts normal. Thanks to the injection of the terminal portion of the hepatic duct, it is possible to observe that the cystic duct empties on its left border.

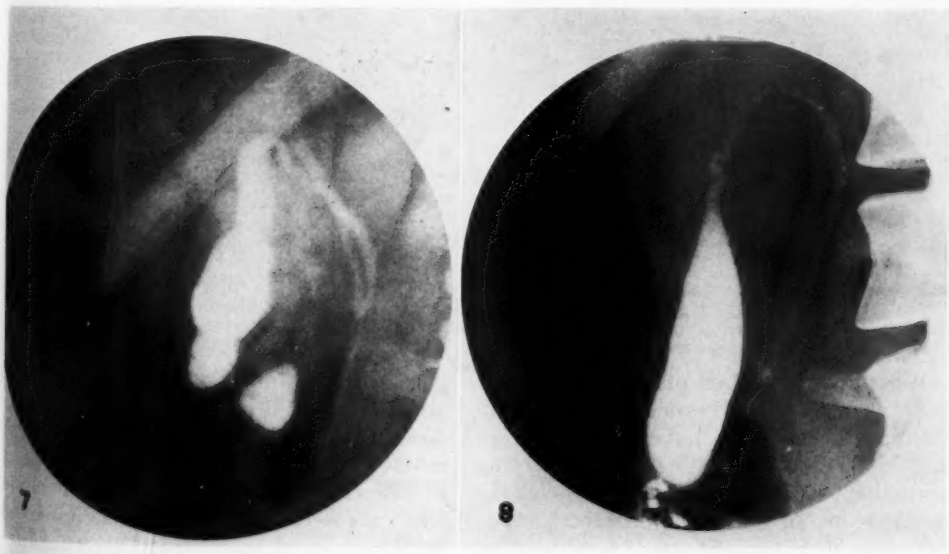
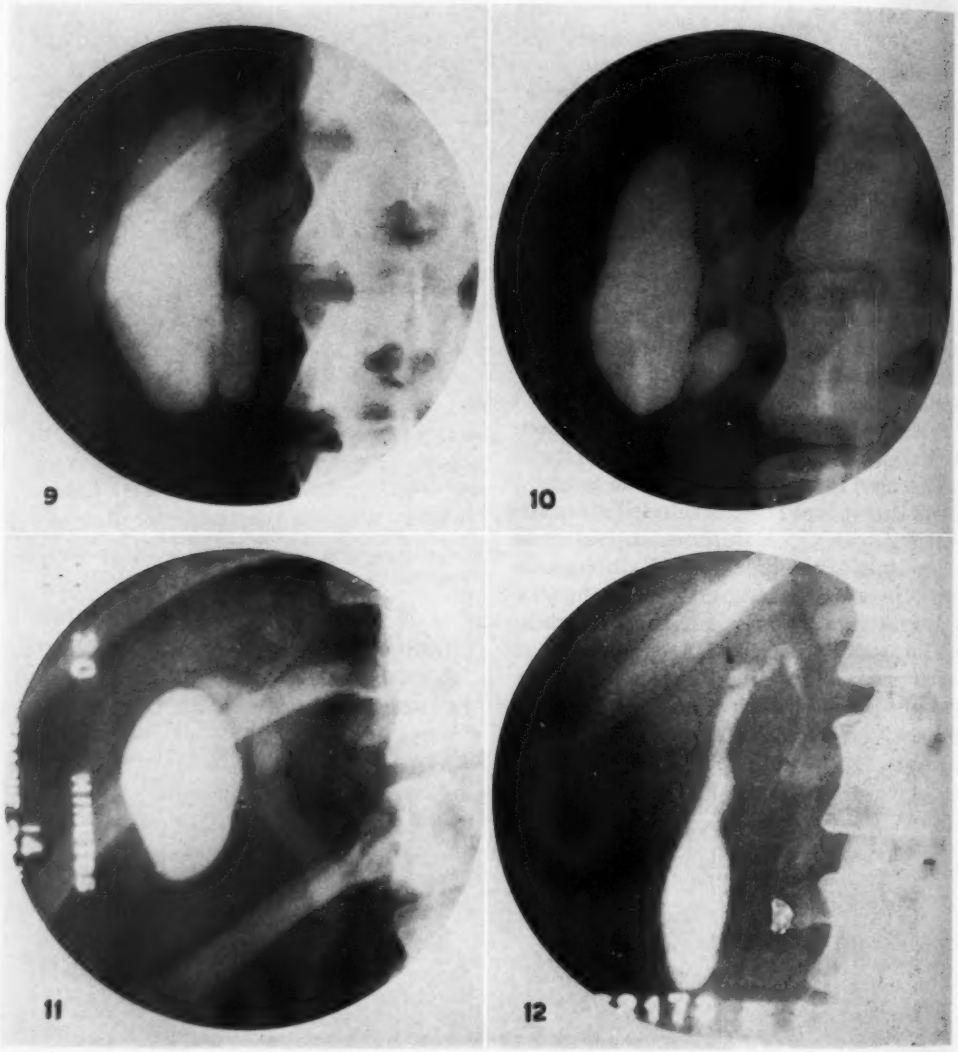


Fig. 7. Telepaque. Gallbladder and ducts normal. Calculus of the right kidney. The cystic duct empties on the left border of the hepatic. Note that the cystic duct is very long and ends very low.

Fig. 8. Telepaque. Gallbladder and ducts normal. The hepatic duct has not been visualized, but the extreme length of the cystic duct makes one suspect that it opens on the left border of the hepatic.



Figs. 9 and 10. Telepaque. Diverticulum of the gallbladder (two views of the same case).
Fig. 11. Telepaque. Diverticulum of the choledochus.
Fig. 12. Telepaque. Diverticulum of the cystic duct.

Finally we would emphasize the clinical value of a roentgen examination in which the gallbladder may have good function and concentration, filling and emptying normally, while the extrahepatic biliary passages are of normal appearance. It is exceptional that symptoms of a gallbladder lesion occur under these circumstances. In the group of patients examined we wish to call special attention to 1 case in which good visualization of the gallbladder was obtained but in which that organ was dilated and emptied with great difficulty. On surgical intervention there was found a filiform cystic duct, and histologic study showed the existence of cholesterosis. In the cases of gallbladder diverticulosis, and in some cases of cholesterosis, in which concentration is good, the diagnosis can be made from the roentgen aspect of the

TABLE VIII: COMPARISON OF AUTHOR'S RESULTS AND THOSE OBTAINED WITH TELEPAQUE ALONE (WHITEHOUSE AND MARTIN) AND WITH CHOLEGRAFIN ALONE (HORNYKIEWYTSCH AND STENDER)

	Per cent
Positive results with Telepaque	
Whitehouse and Martin(500 cases)	90.60
Esguerra-Gómez(1,332 cases)	90.24
Positive results with Cholegrafin	
Hornykiewytsch and Stender(1,000 cases)	96.00
Positive results with Cholegrafin and/or Telepaque	
Esguerra-Gómez(1,400 cases)	98.07

gallbladder. In this connection we wish to quote the valuable opinions of Drs. Bockus (27) and Martin (28) expressed in personal communications in reply to our query as to whether they believed that cholecystectomy was sometimes justified even though the gallbladder was radiologically normal.

Said Doctor Bockus:

"I think it is fair to say that a gallbladder which is normal in every way radiologically, such as you have described, is not likely to be the cause of symptoms. Surely there are rare instances in which some disease may exist in a gallbladder which is radiologically normal. We have this experience occasionally in association with cholesterosis. We occasionally find sand and perhaps very minute calculi are missed with the ordinary technique; these instances must be exceedingly rare.

"If the patient has symptoms which are very characteristic of episodic biliary colic, and the radiologic study is negative, including good visualization of the choledochus, then we usually do a diagnostic biliary drainage. In instances in which cholesterol crystals and/or calcium bilirubinate pigment are recovered and identified without question, I believe that an operative procedure would be justified."

Doctor Martin wrote:

"As I look upon the problem of gallbladder disease from the point of view of a clinician, I believe that most frequently a gallbladder series utilizing Telepaque or some corresponding medium gives accurate interpretation of the function of the gallbladder. I think that generally a well trained clinician suspects gallbladder disease from the history and physical examination and that X-ray findings are thus usually confirmatory. I am inclined to believe that physicians who operate upon patients for postulated gallbladder disease in the presence of a normal roentgenological gallbladder series do so to the disadvantage of the patient, unless the evidence of gallbladder disease is clinically unescapable. The greatest mistakes that are made are in confusing symptoms arising from a psychogenic basis, particularly such situations as abdominal migraine, with the symptoms arising from organic gallbladder disease. From my own point of view, I would be very loathe to operate upon a patient whose gallbladder series and the repetition of gallbladder series showed normal function and no evidence of gallstones."

The opinions which we have just cited emphasize the importance of the negative radiologic examination of the gallbladder and biliary passages in the exclusion of a pathologic state of these organs.

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Cholecysto-Cholangiographia a Excretion: Un Studio Basate Super 1.400 Casos Examinate con Telepaque e/o con Cholegrafin

Le autores reporta un serie de 1.400 studios cholecysto-cholangiographic con Telepaque e Cholegrafin. In 1.218 casos le examine initial con Telepaque esseva adequate e Cholegrafin non esseva usate. In 114 casos ambe medios esseva empleate. Cholegrafin produceva melior resultatos in patientes cholecystectomisate e in patientes con non-function del vesica biliari de longe duration, e illo esseva usate sol in 68 tal casos.

Resultatos positive esseva obtenite in 1.202 del 1.332 casos examinate con Telepaque sol (833 normal, 369 pathologic). Isto es ben de accordo con le resultatos de Whitehouse e Martin qui obteneva resultatos positive in 90,6 pro cento de 500 casos examinate con Telepaque. Pro le serie total, le procentage de observationes positive esseva 98,07, in comparation con 96 pro cento obtenite per Hornykiewytsch e Stender in 1.000 casos in que Cholegrafin esseva usate sol.

Le autores sublinea le importantia de negative examines radiologic pro le exclusion de morbo de via biliari.

Hypaque, a Safe Medium for Pediatric Intravenous Urography[1]

RICHARD F. McCLURE, M.D.

MANY REPORTS have appeared in the literature concerning a new intravenous contrast medium, Hypaque. The usefulness of the drug in examination of the adult is well known. This report concerns its use in the pediatric patient. It summarizes briefly experience with the drug in the study of 226 patients at the Children's Hospital Society of Los Angeles, Calif. The report is limited solely to the use of the drug in intravenous urography. The youngest patient in the series was two weeks old, and the oldest fifteen years.

PROCEDURE

Patients under five years of age were prepared simply with enemata and an eight to twelve hours fast. Those over five years of age also received catharsis, usually by castor oil in appropriate dosage for size and age. Only infants were not fasted or dehydrated. Carbonated beverages were used frequently in infant examinations after the manner of Hope and Campoy (1).

Skin tests were performed in each of the patients referred for intravenous urography. Positive reactions (a wheal surrounded by an area of erythema at the site of intradermal injection of 0.5 c.c. of Hypaque) were found in 16 patients. Seven of these positive skin reactors were under twelve months of age, and the remaining 9 were distributed evenly over the older age groups. One of the 9 exhibited urticaria. The examination was cancelled in all of the positive skin reactors.

All of the 210 negative reactors received Hypaque intravenously according to the following schedule:

Under 6 months of age	8 c.c.
6 months through 11 months of age	10 to 13 c.c.
12 months through 36 months of age	14 to 18 c.c.
Over 36 months of age	20 c.c.

In those groups where a range of dosage is stated, the younger patients received the smallest amount and the oldest the largest amount, with intermediate dosage according to age. All intravenous injections were given over a period of three minutes. Films were made at three, five, and seven minutes after completion of the injection. Additional films were obtained as indicated following study of the first three films.

RESULTS

The opacification of the urinary tract in all cases was of uniformly good quality. The intrarenal structures and the upper two-thirds of the ureters were satisfactorily visualized. The lower thirds of the ureters were not invariably seen on all films in a single examination but usually could be adequately demonstrated on at least one of the films in the series. The urinary bladder was always well shown. When an excessive amount of intestinal gas obscured the ureters, a lateral film revealed them adequately.

REACTIONS

Among the 210 patients who received a large amount of Hypaque intravenously, only 4 experienced nausea and vomiting. Four additional children were nauseated but did not vomit. All of these 8 patients were over six years of age.

Such signs as urticaria, wheezing, choking, and pallor, which are quite definite evidence of drug intolerance in urography, were not observed in any of the cases in which a full dose of the drug was given. In the infants and very young children, who were obviously frightened and often struggling violently in resistance to the procedure, flushing, sweating, and increased pulse rate were observed. But these signs disappeared promptly following re-

[1] From the Department of Radiology of the Los Angeles Children's Hospital, and the University of Southern California School of Medicine. Accepted for publication in December 1956.

moval of the needle from the vein. We do not consider them as reactions to the Hypaque.

CONCLUSIONS

When used in the manner described in this report, Hypaque is a most satisfactory contrast medium for pediatric intravenous urography. Opacification of the urinary tract is quite satisfactory. Positive skin reactions contraindicating the examination were few. Adverse reactions to the intravenous administration of the full dose of the medium were few and minor.

NOTE: The Hypaque used in this study was generously donated by Winthrop Laboratories.

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SUMMARIO IN INTERLINGUA

Hypaque, un Salve Substantia de Contrasto pro Urographia Intravenose in Patientes Pediatric

Hypaque se ha provate un salve e utile substantia de contrasto in le execution de urographia intravenose in infantes e juveniles. Le serie hic reportate consisteva de 226 patientes de etates de inter duo septimanas e dece-cinque annos. Cutireactiones esseva obtenite in omne casos. Dece-sex habeva reactiones positive, e in iste casos le proponite examine non esseva effectuate. Le remanente 210 patientes recipeva injectiones del substantia in doses variante ab 8 cm³ pro subjectos de minus que sex menses de etate a 20 cm³ pro subjectos de plus que tres annos. Le opacification del vias urinari esseva uniformemente de bon qualitate in omne le casos. Solmente alicun reactiones minor esseva notate.

Para-Articular Ossification, A Complication of Anterior Poliomyelitis.

A Case Report[1]

LAWRENCE L. LARSEN, M.D.,[2] and HOBART H. WRIGHT, M.D.[3]

THE DEVELOPMENT of para-articular ossifications associated with anterior poliomyelitis is considered an unusual complication, although para-articular calcification and ossification are not infrequent sequelae of traumatic paraplegias or cord lesions with accompanying sensory deficits.

Drehmann (1) in 1927 reported a case of para-articular calcification following acute anterior poliomyelitis in a boy two and a half years old. Three cases of myositis ossificans, all in adults, were published in 1951 by Costello and Brown (2). In 1952 Freiberg (3) reported an unusually extensive case of calcification and ossification of hips, shoulders, and metacarpophalangeal joints, and cited 2 other cases, in adults, with extensive calcifications in the hip regions.

The authors recently observed an extensive ossification of the left hip in a patient in the convalescent stage of acute anterior poliomyelitis. This complication seems not to have been reported previously in the roentgen literature, except as noted by Caffey (4).

B. R., a 12-year-old white girl, was admitted to South View Isolation Hospital on Sept. 10, 1955, with poliomyelitis, confirmed by laboratory findings. The immediate hospital course was one of progressive weakness of all extremities, most pronounced in the right arm and left leg.

After an initial ten-day isolation period, the patient was transferred to St. Luke's Hospitals, where, in addition to the above findings, numbness of the left anterior thigh was noted. The prescribed post-polio treatments were given during the succeeding three months, but the response was not as anticipated, in that posture became progressively poorer as a result of contractures of the left hip.

A careful review of the family and personal history revealed no evidence of local injury or disease of the left hip, or of previous significant illness.

Fig. 1. Appearance of the left hip four months after the onset of poliomyelitis.

Preparatory to surgery for correction of contractures of the hip, roentgen studies were done on Jan. 6, 1956. These revealed an extensive para-articular ossification (Fig. 1). Laminagraphy of the involved area failed to reveal any evidence of fracture or intrinsic disease in the femur. Complete skeletal surveys showed no similar involvement elsewhere. Relevant laboratory findings were: serum total protein, 7.3 gm. per cent; serum phosphorus, 4.6 mg. per cent; serum calcium, 10.4 mg. per cent; alkaline phosphatase, 7.4 Bodansky units.

Follow-up x-ray studies during the succeeding six months revealed findings consistent with bone of increasing maturity (Fig. 2).

Surgical extirpation was considered advisable

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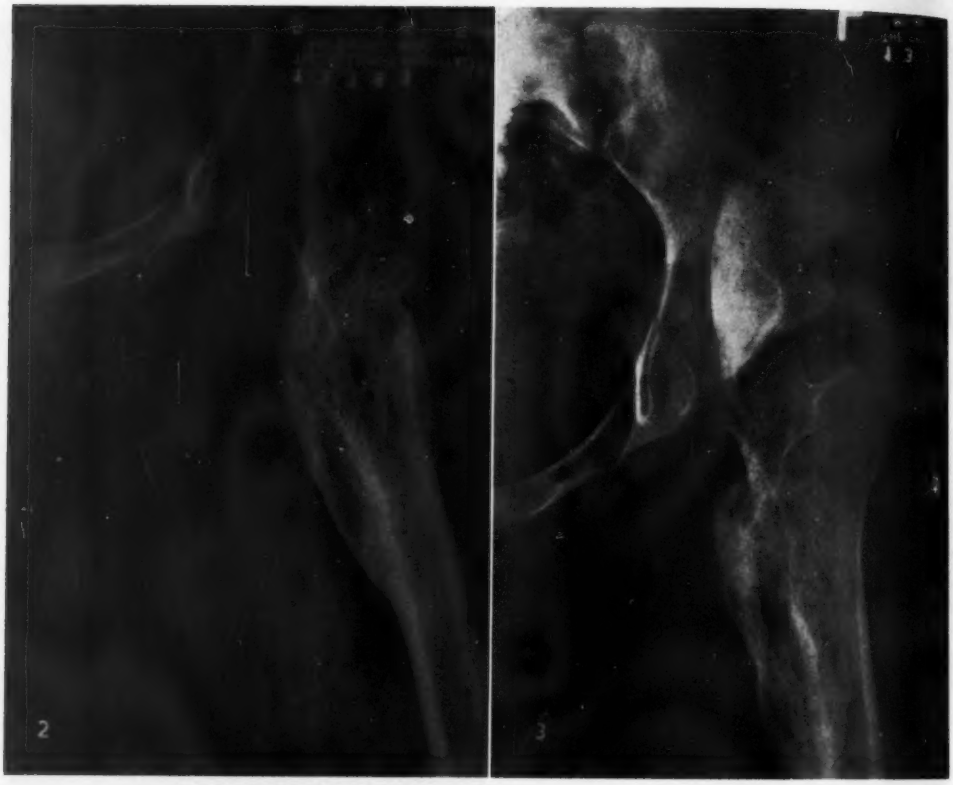

Fig. 2. Bone of increasing maturity nine months after onset of disease.
Fig. 3. Immediate postoperative roentgen appearance.

because of the extreme flexion contractures and fixation of the hip. Operation was performed on June 29, 1956, ten months after onset of the disease. The major muscle groups were identified and found not to be replaced by ossifying tissue. An extensive formation of mature bone was seen arising from the anteromedial aspect of the upper femur and extending to the joint capsule and the ilium. Complete excision at this time was not considered possible, because of involvement of the neurovascular bundle distally. Free range of motion was restored to the joint by removing large portions of bone down to the joint capsule (Fig. 3). Gross and microscopic examination resulted in a diagnosis of reactive bone formation. No muscle or other connective-tissue ossification was present.

A program of physical therapy was resumed approximately two weeks postoperatively. The patient exhibited marked improvement in muscle strength and was discharged from the hospital on Aug. 3, 1956, to be followed as an out-patient. She became completely independent at home and no longer required a wheel chair, although crutches were needed in walking. Further surgery to acquire even greater motion may be necessary.

DISCUSSION

Several theories have been postulated as to the mechanism of ossification. Tissue atrophy, degeneration, or metaplasia remain unproved. No new theory is here advanced. Metabolic studies were not made during the early convalescent period in the reported case, but later studies appeared to rule out inherent defects of bone metabolism. It is thought that the well planned and executed physical therapeutic measures did not contribute to the complication. The associated sensory involvement, as noted early in the course of the disease, may have been of some etiologic importance. Perhaps the infrequency with which this complication has been reported may be attributed to the fact that only occasionally are roentgenographic examinations of poliomyelitis patients obtained. When ossification is discovered

early in the course of convalescence and found to be minimal, roentgen therapy might be considered.

SUMMARY

A case of para-articular ossification complicating anterior poliomyelitis in a twelve-year-old girl is presented. The etiology remains obscure.

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SUMMARIO IN INTERLINGUA

Ossification Para-Articular, un Complication de Poliomyelitis Anterior

Le autores presenta un caso de ossification para-articular del coxa sinistre in un puera de 12 annos de etate, occurrente post poliomyelitis anterior. Dece menses post le declaration del morbo, un extense formation de osso matur, saliente ab le aspecto anteroposterior del femore superior e involvente le capsula articular e le ilio, esseva partialmente abferite. Un excision complete non esseva possible, sed un libere latitude de movimento esseva restaurate in le articulation. Le patiente habeva devenite completemente independente in su domicilio e non requireva plus un chaise rolante. In ambular, il es ver, illa usava crucias. Esseva opinate que forsan un secunde operation esseva ne cessari.

Isotope Circulation Studies in Congenital Heart Disease

RICHARD H. GREENSPAN, M.D.,[2]
RICHARD G. LESTER, M.D., and
JAMES F. MARVIN, Ph.D.

A new method for the detection of intracardiac and extracardiac shunts in congenital heart disease has been developed, offering several advantages as compared with conventional dye-dilution curves and obviating the necessity for arterial puncture. The new procedure is simple to perform and promises to yield additional direct information concerning the pulmonary and intracardiac circulation which, with dye-dilution methods, can be obtained only by inference.

Ten to one hundred microcuries of radioiodinated serum albumin are injected either intravenously or into cardiac chambers at time of catheterization.[3] Externally placed collimated scintillation counters are positioned over the heart and femoral artery. It is planned to place still another counter over the peripheral lung field, and in certain instances it may become desirable to put separate counters over the arterial supply to the upper and lower extremities. Initially, the recording apparatus consisted of a Berkeley Computing Rate Meter and an Esterline Angus recorder. With this apparatus we were unable to record several simultaneous curves, so that separate indicators were used for injection time and appearance of the isotope within the heart. (The latter was recorded by a single impulse obtained when 100 counts had accumulated from the counter placed over the precordium.) Because of several inherent disadvantages in this system, a transistorized integrator was developed by one of us (J. F. M.). This, along with a four-channel Sanborn Polyviso recorder, enables us to obtain simultaneous curves from our counters and electrocardiographic tracings.

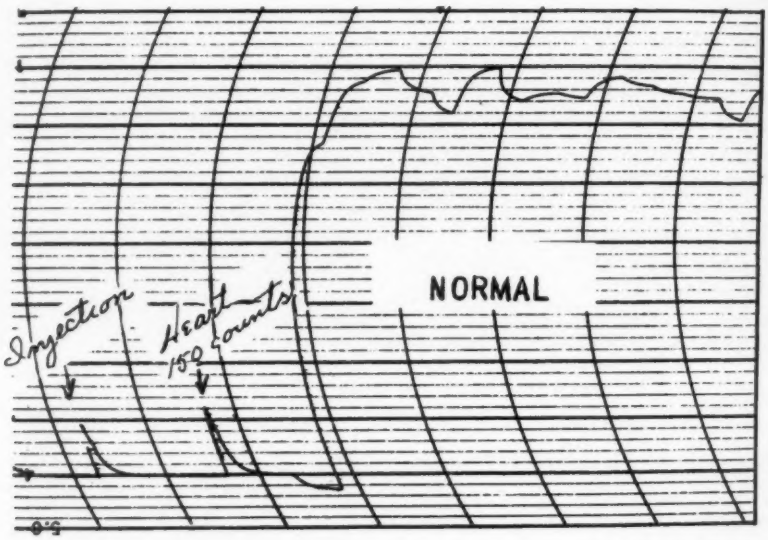

Fig. 1. Study on a normal dog, utilizing Berkeley Computer and Esterline Angus.

Preliminary work was carried out on normal dogs and on animals with experimentally produced shunts. Following this, studies were made on patients with a variety of congenital defects as well as on normal subjects.

When the injection is made into the heart or great vessels at the time of catheterization, the counter over the heart records the radioactive material as a large bolus. If injection is made into the pulmonary artery or a peripheral vein, the bolus effect is not as sharp. In normal subjects, the collimated counter over the femoral artery then observes a precipitous rise, followed by a slight decrease in activity before equilibrium is reached (Fig. 1). At the time of the

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[2] Fellow, American Cancer Society.

[3] It is planned to use I^{131}-labeled contrast material to facilitate rapid excretion of radioactivity.

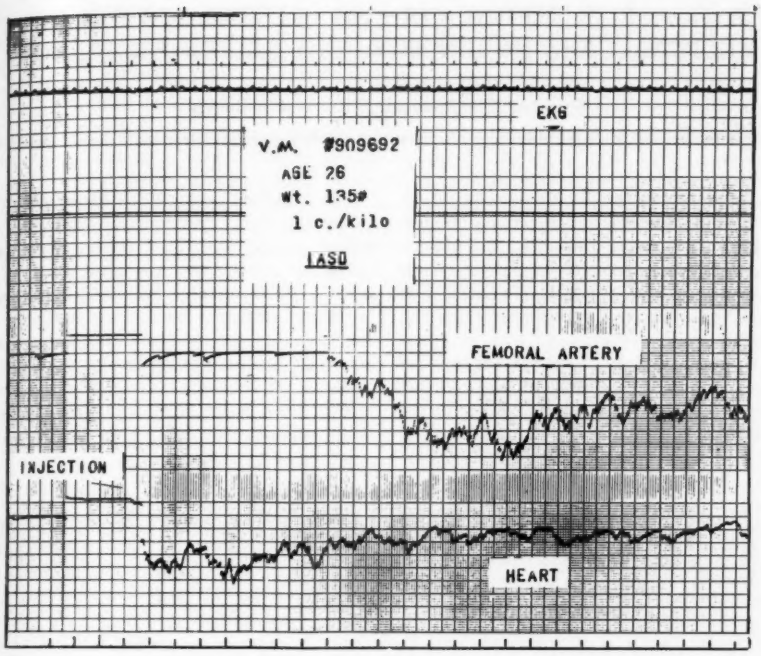

Fig. 2. Left-to-right shunt showing more gradual slope of femoral artery counts and evidence of heart recirculation. Utilizing transistorized rate meter.

femoral artery rise, counts over the heart have decreased.

In left-to-right shunts the pattern is quite different. The time of appearance of radioactivity over the femoral artery is the same as in normals, but the slope of appearance is much more gradual, and the precordial counter records continuing high activity, indicating recirculation (Fig. 2). It is expected that a counter placed over the peripheral lung field will add further confirmation of recirculation through the pulmonary vascular bed.

Right-to-left shunts are characterized by very rapid appearance of varying concentrations of radioactivity over the femoral artery (depending on shunt size), with the cardiac record depending on the abnormal hemodynamics produced by the particular malformation. Location of shunt is accomplished by serial injection of chambers and pulmonary artery.

Previous work has been done with counters placed over the heart, and femoral puncture has been used to determine flow rates and concentrations of isotope (1–6). These studies have yielded valuable data regarding cardiac output and various abnormalities of the heart. We feel, however, that the method presented will yield considerably more complete and precise information. Further studies and refinements of technic are being carried out.

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A COLLEGE REPORT ON THE PRESENT STATUS OF MEDICAL CARE INSURANCE AND ALLIED PROBLEMS[1]

WILBUR BAILEY, M.D.
President, The American College of Radiology

A GOOD ALTERNATIVE title for this presentation would be "The College Views the Calculated Risk," for there are other risks of importance just now besides those covered by medical care insurance. Recently, for example, the Executive Committee of the College had a press conference in New York with leading science writers of newspapers and magazines in an effort to suggest accurate sources of information and to prevent further unfortunate interpretation of last June's National Academy of Science report, published in booklet form under the title "The Biological Effects of Radiation: Summary Reports," and simultaneously released as a "Report to the Public." Six subcommittees of the Academy reported on the effects and danger of atomic radiation in the field of genetics, pathology, meteorology, oceanography and fisheries, agriculture and food supplies, as well as medical and dental uses of x-rays. Only the medical and dental uses and the possibility of H-bomb fall-out seem to have made "scare headlines."

As is the usual custom with reports made by x-ray specialists to physicians who consult them, the Committee made a division between the objective findings and the conclusions which might be derived therefrom. With the objective findings of most of the distinguished scientists on the Committee, we have little quarrel, although many of us feel that the genetic experiments on fruit flies, guinea-pigs, rabbits, or other lower animals, can be compared with but limited scientific accuracy to the effect x-ray exposure may have on the human race.

Yet to be untangled in the public mind is the fact that much of the report deals with the dangers of radiation to the entire population from fall-out. Present levels of all radioactive fall-out substances are far below the danger point and, while we must be ever alert in this new atomic age, some persons may find it reassuring to know that the maximum effects of the present overall radiation of the fall-out type are not to be expected for another fifty generations, which is to say about a thousand years from now.

Although the public has in many cases not grasped the distinction, we continue to try to make it clear that fall-out radiation is something very different from x-ray examinations by properly trained doctors using appropriately shielded equipment, involving small areas of the body.

The National Committee on Radiation Protection, under the able direction of Lauriston Taylor, has issued handbooks in the last quarter century on radiation protection, the sum total of which is almost 2 inches thick. Further, we make every effort to teach medical students as well as our fellow practitioners to treat radiant energy with great respect. In the meanwhile, our some five thousand radiologists continue to be guided by the principle which they have always observed, namely, "the minimum necessary exposure," in making medical x-ray examinations.

However, we have now to consider not only our own relatively small group of professional radiologists, but other physicians and surgeons, dentists, and veterinarians, bringing the total number of users of x-rays to over 100,000 plus an equal number in industry. The College proposes to continue its educational efforts, and welcomes sincere and informed advice about radiation protection from any quarter.

The risks in the medical use of x-rays are calculated ones and statistically are not great. There is no doubt of the value of examination of the pregnant woman, under proper indications, to determine the size of the pelvis, from which one can calculate whether the baby's head is so large that the "passenger will not fit the passage." There are more than 160,000 spastic children in the United States, many of whom owe their brain damage to prolonged labor in a narrow pelvis—a situation which might have been predicted and avoided beforehand by calculations made from x-ray films. The amount of radiation used in making such measurements has never been proved to be dangerous, but many spastic children exist as proof of the very definite danger of prolonged birth canal pressure on an unborn infant's brain.

Another calculated risk is the purposeful over-irradiation of an area of skin in the treatment of underlying cancer. On one side of the ledger is the certainty that the patient will die if the cancer is not sterilized by sufficient irradiation. If the cancer is thoroughly treated, the patient does not die, and reparative skin grafting operations are minor and simple.

We are, therefore, all in general agreement that the medical use of x-rays should be continued by doctors who know the inherent risks. There is debate, however, whether too frequent free chest

[1] Presented at the Forty-second Annual Meeting of the Radiological Society of North America, Chicago, Ill., Dec. 2–7, 1956.

examinations on seemingly healthy people—"come in whenever you feel like it"—are entirely in the public welfare. Surely, fluoroscopy to determine whether or not a shoe fits can have dangerous potentialities.

As of the present moment, to show the need for eternal vigilance, along comes *Life* magazine with a laudable article on "How to Interest Young People in Science." Unfortunately, one of the do-it-yourself experiments involves making a home-made x-ray machine. For $2.50, it seems, one can get a Ford spark coil, a tube of some sort, which appears in the illustration to be shielded by a tin can, and thereby develop x-rays in sufficient amount to show the bones in a hamster and incidentally, though this is not mentioned, to burn the fingers off the boy experimenter.

While still on the subject of hazards, we may mention one of the greatest concerns at the moment, namely, what might be called the "restrictive labeling of drugs." As Doctor Isadore Ravdin, President of the American College of Surgery, recently pointed out at a San Francisco meeting, pharmaceutical houses are now telling doctors how to practice medicine. Nowhere could this be more true than in the field of the intravenous preparations which we as radiologists use to portray the interior of the urinary tract, the biliary tract, and the heart and great vessels.

These drugs do sometimes provoke reactions, and it is true that about once in 100,000 injections a death, or serious reaction, will occur from some completely unpredictable idiosyncrasy. This figure from a statistical standpoint is minuscule, compared, for example, to the fact that in Los Angeles County one automobile driver in thirteen may expect a serious accident every year. The patient's relatives, however, even as you and I, tend to overlook the fact that complications and idiosyncrasies are as inevitable in medicine as in every other field of life. Quite naturally they look for someone to blame.

"Instructions" issued by the manufacturers frequently mention the necessity for a preliminary test with a small amount of the drug in question. Actually, there is a general agreement among medical men that preliminary tests are worthless, and that, in the rare cases of extremely sensitive patients, small amounts are as deadly as larger ones. Then come further "instructions," to the effect that five or ten minutes should be taken to inject 20 c.c. of a 50 per cent solution, though in actual practice two and a half times that amount of a 70 per cent solution must be injected in two or three seconds if films of satisfactory diagnostic quality are to be obtained in some procedures.

We doctors know these things, but the public does not. Not long ago, for example, a judge in San Francisco, in his instructions to the jury, pointed out that a doctor was negligent because, even though he had followed the custom generally used in the community, he had not observed the directions accompanying the bottle. Result: A verdict for $250,000 against the doctor.

The situation is far from hopeless, however, as we have found from a discussion with representatives of the Food and Drug Administration. To begin with, we may expect manufacturers to revise their labeling to conform with modern practice. Much more important would be a change in the tone of the instructions, so that rather than "commands" they would merely be "suggestions for use." Equally important, the label should indicate that the medical judgment of the physician is always to be considered as paramount to any suggestions from the manufacturer.

To turn to the question of medical insurance and some allied problems: Nothing is more apparent than the fact that changes are going on in this field—changes, indeed, in which the American College of Radiology has had great influence.

Within the past few months, nearly a million CIO steelworkers and their dependents have come under a vast medical and hospital insurance effort which combines Blue Shield, Blue Cross, and some commercial companies in a contract which for the most part is to our liking. Within this framework of management-union activity, which can rock the ship of American economy, the negotiators at their collective bargaining tables have taken sufficient time to look at medical radiology and place it in its proper role: medical service.

We continue to insist that radiology *is* a medical service, not a hospital function. Possibly there are those among you who wonder why. That radiology has not always been considered the "practice of medicine," is indicated by the fact that only within the past three years have some ten to twelve state Blue Shield Plans finally managed to bring the medical efforts of the radiologist into Blue Shield. Right now there are forty-six Blue Shield Plans which have in-hospital diagnostic coverage, and fifty such plans with ambulatory patient coverage. Recent changes across the country, most of them favorable, are too numerous to mention.

In short, this question of change from Blue Cross, a hospital plan, to Blue Shield, the doctor's plan, is one of the basic issues in medical economics today, and the College of Radiology is making a constant and continuous effort to bring about this conversion, as well as to have Blue Shield cover office radiology done by a doctor who limits his practice to radiology and to whom patients are referred. Such a plan has the advantage from the patient's standpoint of providing a consultation, while from the economic standpoint it tends to prevent overutilization.

Such improvements are not made without effort, and the continuing state of flux which besets medical economics is not without its drearier moments.

For instance, only a few short months ago Blue Cross jumped with both feet into medical insurance. The scene was Pittsburgh, Penna., and the doctors in that area—all the doctors, not just the radiologists—did something about it. In answer to the threat, the Blue Shield Plan in Pennsylvania has now developed new diagnostic x-ray services, in or out of a hospital, which will be offered to subscribers in certain groups in the area affected by the Blue Cross announcement. In Massachusetts a similar plan, beneficial to patients and radiologists, is now in effect.

Let me here touch briefly on another economic Goliath. This is the Defense Department's "Medicare Program," the hospital and medical insurance coverage for members of the Armed and Uniformed Services of the United States and their dependents. This enormous undertaking has been closely coordinated with state medical societies, and through liaison with the American Medical Association, among others. Details are being worked out on a state by state basis. It behooves us all to keep alert, to be vigilant, and to serve as members on Medicare boards of consultants.

We are pleased that radiology has been clearly defined by the recent Iowa decision as the practice of medicine, and not simply a specialty to be covered by hospitals and technicians. But we cherish no illusions that hospital administrators and business men elsewhere will not try to exploit radiologists for their personal profit. The commissions and committees of the College, as many of you who serve on them are well aware, are constantly active, and they should be, for many of the schemes which are being repeatedly proposed by business administrators are not in the best interests of the patient. It is our sincere belief that the patient's welfare must be paramount. Otherwise we would never have started on the arduous and lengthy road of education which eventually led to the practice of medicine.

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EDITORIAL

Genetic Injuries

"Race hygiene" should be an idealistic concept, projecting our finest ambitions into the future of mankind. Under Hitler the term became an instrument of propaganda, a cloak for antisemitism. Since publication by the National Academy of Sciences of its *Summary Report on Genetic Injuries from Radiation*, we are seeing a renewed interest in race hygiene. We should take care lest it develop into a movement against radiologists. It would be too bad if the small threat of radiation injury should lead people to deny themselves the large benefits of diagnostic radiology.

After a television broadcast on radiation injury, one thoughtful viewer commented: "You're making people afraid to have an x-ray." After years of listening to the phrase "the x-ray shows . . . ," this would indeed be amazing. But, on a cooler view, one must feel that no harm would come from an inclination to estimate the expected value of an x-ray examination versus its chance of harm. The physician would have to defend his judgment in prescribing it. The radiologist would have to reassure himself and his patient that he can do it efficiently and safely. Both these self-inspections could be wholesome.

Popular reaction to the genetics *Report* does appear at times to verge on the allergic. Scientists as well as lay writers are slow to achieve a sense of balance. This idea of the harmful effect of even the smallest amount of radiation is hard to get understood properly. So many people have difficulty with quantitative concepts. Their world is all black and white—no more half-tones than a VIP cartoon. Moreover, the measures in radiology and genetics are unfamiliar, running to unobservable dimensions and astronomical numbers. One doubts if all the writers and commentators have understood the words they were using. One even doubts, in some cases, if they have read the *Report*. Much good could be achieved, surely, if people could be persuaded to read it before discussing it. It presents most admirably the scientists' ideals and care in examining the problem and arriving at conclusions on the basis of what information can be had, allowing for the things we do not yet know as precisely as we would like.

Radiologists have two good reasons for being informed. First, they have to design their technics to minimize radiation hazard. Second, they can expect their clients (physicians) and their patients to be asking questions.

Radiation is indeed injurious. Radiologists all have first-hand acquaintance with its harmful as well as its benign effects. They regularly produce severe radiation injuries, just as the surgeon produces severe wounds in the treatment of malignant tumors. Small amounts of radiation do constitutional harm that may not emerge as visible injury for twenty years. Often quoted is the statistic about leukemia—ten times as frequent among radiologists as among physicians in general. If this is due to over-irradiation, it dates from a time when radiologists were less aware and less careful about stray radiation. Often referred to, also, is the "shortening of the life span" from small doses of radiation. Well demonstrated in small animals, it is practically out of reach for experimental measurement in man. The favorite quotation is Hardin Jones' speculation that every roentgen of total-body exposure shortens life by ten to fifteen days. Supposing there is some truth in this, there is a better way to put

it: Even small exposures tend to age a person prematurely, just as do repeated infections and other vicissitudes of life. People in a healthy society are physiologically younger at sixty than those in a disease-ridden society are at fifty.

In general, radiation injuries heal, as other injuries do, leaving some scars and sequelae, including the tendency to aging and also a measurable tendency to carcinogenesis. These things are to be understood quantitatively, with appreciation of the fact that the harm done by small amounts of radiation is out of reach of direct observation. The internationally accepted maximum permissible exposure of 0.3 r per week is based on the conviction that it will lead to no perceptible injury.

From genetic injuries, by which we mean the induction of heritable mutations, there appears to be no recovery. Every mutation that occurs persists, generation after generation, until removed by the chance failure of the particular line of inheritance by the death of the person(s) carrying the mutation before he has had any children. (This includes extrusion of the mutant chromosomes into a polar body in maturation of the ovum, and the corresponding segregation in maturation of sperm.) Mutations are occurring all the time. Of the individually recognized mutations, the rate is from 5 to 100 per million per generation. Some of these are incompatible with childbearing and die out in the first generation. Others have so little effect on survival that it takes many generations to eliminate them. The accumulated pool of deleterious mutations must number several thousand in any large population and has been estimated at eight in every matured ovum or sperm (on the average).

Of the natural (spontaneous) mutations, only a small fraction are caused by radiation. In consequence, the gonadal exposure required to double the natural mutation rate is many times the natural exposure rate from cosmic and terrestrial radiation (less than 0.1 r per year in most places). The yearly dose from radioactive potassium, carbon, and radium in the human body amounts to only a quarter as much. The "doubling dose" of radiation (the amount that would make mutations occur twice as frequently) appears to be only about one-tenth as much for mammals as for insects and has been estimated variously as between 15 r and 150 r.

The Genetics Committee of the National Academy of Sciences advised keeping records of every person's exposure to radiation and limiting the total to 30 r by the age of thirty, when the majority of children will have been conceived. This looks to be an impossible program. Moreover, the genetic harm depends not on the occasional high individual exposure, but on the average. The aim should be to limit the exposure totaled over the 150 million of our population. In large populations statistical calculations become quite dependable and we can deal confidently with the average exposures and stop worrying about the occasional bad chance. It is usual to take pride in "leaning over backward" when figuring radiation hazard. In this case, however, the only proper attitude is bolt upright. If radiation escapes out onto the sidewalk, one properly multiplies the milliroentgens per day by the average number of persons there; one doesn't fret about the possibility that a newsboy will stand there for six hours—that is, as far as genetic injuries are concerned.

In regard to personal injury, the present permissible limit of 0.3 r per week is not resulting in any injuries that can be detected. We may be suspicious about leukemia, but we don't know the exposures that have induced it in radiologists (if it has been so induced). There is an inclination to stretch out the time over which the limiting exposure may be averaged, so that a brief exposure rate above 0.3 r per week will be permissible if the total in a year does not go over 5 r. If one considers the fraction of the population industrially exposed to radiation, with generous prediction of the numbers that will soon be employed in nuclear

power plants, etc., it is easy to see that the present industrial exposure limit of 0.3 r per week, even if integrated to 15 r per year, still leaves the average exposure of the entire population very low. The usual demand is that non-industrial exposure be limited to less than one-tenth of what is permissible to persons employed where they know the hazard. This low general exposure rate will be well assured if we live up to the present international limitation of 0.3 r per week for employed persons.

The genetics *Report* and the corresponding report of the British Medical Research Council[1] bring out some disturbing figures in regard to medical radiology. The number of diagnostic radiographs is so large that the small doses add up to a surprising total. At present, the use of x-rays for medical diagnosis is far and away the most important man-made irradiation hazard from a genetic standpoint. The British report says that it adds 22 per cent to the natural (cosmic and terrestrial) irradiation. Estimates for the United States indicated an added gonadal dose about equal to the natural irradiation. Compare this with the general average from occupational exposure and the present contribution from nuclear bomb testing, which together add only a few per cent to the natural level! It is true that fallout from the bombs is adding strontium 90 and caesium 137 to our diets (mostly in milk) in measurable amounts. Cesium 90 gamma rays now show up as about half as much as the irradiation due to the body's normal content of potassium 40.

If we can think quantitatively and logically, not emotionally, we shall see that the present genetic hazard is not atom bombs nor nuclear energy, but x-rays, particularly as used for medical diagnosis. Let us not condemn roentgen diagnosis, for it yields incomparably more health than it destroys. Let us not condemn minifilm chest surveys, for the gonadal dose is an insignificant percentage of the total. The same can be said of dental radiography. The place to put our attention is the lumbar spine, abdomen (especially pyelography), pelvis, and hips; here extra care on the part of radiologists could cut the potential genetic mutations in half.

We radiologists have long understood the obligation to see that our radiations hurt no one. As the number of persons being irradiated increases, we are having to take on a more stringent duty, namely not to let our radiations hurt the race, even by the accumulation of small doses given to large numbers of people. Thirty roentgens produce the same total chance of a mutation appearing whether given in one dose to one ovary or in doses of 30 microroentgens to each of a million persons living in forty-eight States and irradiated at one time and another over a span of thirty years.

We should be constantly reminding ourselves that a mutation put into the race can be eliminated only by virtue of increasing the chance of death for those persons who carry it. The number of mutations introduced must be balanced by the number deleted by premature death (in some future generation) of carriers of the mutation before they beget children. The less harmful a mutation is, the longer it will be before it is deleted. This affords a mean paradox: The less harmful mutations are the ones that do the most total damage because they persist through so many generations.

The cause will forever be untraceable. The person will die "of natural causes." If identifiable as a metabolic constitutional deficiency, any single case can be attributed to natural "spontaneous" mutation with much greater likelihood than to man-made radiation. The fact that the cause cannot ever be traced does not, however, relieve the physician's (radiologist's) conscience of the responsibility.

The dose to the gonads of every patient (who later begets children) adds onto the gonadal doses of previous patients. Every time the accumulated total reaches 30 r,

[1] The Hazards to Man of Nuclear and Allied Radiations. June, 1956.

you can say: Someday, somewhere, in a future generation unpredictable, the extra mutation probably put into the pool of mutations by these x-ray doses will drop out again by premature death of a person who carries it. We look to see radiologists re-examine their technics and make sure that the gonads are guarded against useless radiation by proper cones or special shields. Familiarity does not breed contempt, but rather increased intelligent concern. R. R. NEWELL, M.D.

Radiologists and Protection from Radiations

The publicity resulting from the development of nuclear power has called the attention of all thinking citizens of the world to the dangers of ionizing radiations. To the public at large and to many scientists the existence of such hazards comes as a great surprise and they treat the problem as if it had never before been considered. In studying the subject, a committee of the National Academy of Sciences found that diagnostic x-ray examinations were one of the greatest sources of exposure of the public to ionizing radiations. Ignoring the fact that such exposures are often the means of saving individual lives and of protecting the public health, and ignoring previous activities of radiologists and their colleagues, the committee called upon the medical profession to *initiate* a movement for protection.

Without going into all the history of efforts at protection, which started in 1896, I would like to point out that in 1920 the American Roentgen Ray Society formed a committee to recommend protection measures, which committee reported in 1922. From the combined efforts of this committee and similar groups in other countries, an International Committee on X-Ray and Radium Protection was formed, which established certain rules and regulations. The original American Roentgen Ray Society committee also served as the nucleus for an Advisory Committee on X-Ray and Radium Protection which functioned under the sponsorship of the National Bureau of Standards but was made up of representatives from the various national societies and manufacturing companies interested in x-rays. In 1946, after World War II, this National Committee recognized that the problems of radiation protection were no longer limited chiefly to medical x-ray installations and therefore expanded its membership to bring in other interested groups. In keeping with this change of emphasis, the name was changed to "The National Committee on Radiation Protection." The committee is made up of representatives of the following organizations:

American College of Radiology
American Dental Association
American Industrial Hygiene Association
American Medical Association
American Radium Society
American Roentgen Ray Society
National Bureau of Standards
National Electrical Manufacturers Association
Radiological Society of North America
U. S. Air Force
U. S. Army
U. S. Atomic Energy Commission
U. S. Navy
U. S. Public Health Service
Three representatives-at-large

There are now so many different scientific disciplines necessary to the work of the Committee that it has formed many subcommittees whose members are selected on the basis of their scientific knowledge. The main Committee is a policy-making body, which also correlates the work of the various committees. For purposes of expediency, an Executive Committee conducts the affairs of the main committee between its meetings, and much work is done by mail.

The National Committee works in close coordination with an International Com-

mission which is appointed by the International Congress of Radiology. In this way, all countries, including Russia, get the benefit of the experiences in all other countries. The subjects considered can best be presented by giving the names of the subcommittees, which indicate the aspects of the protection problem with which they deal:

Permissible Dose from External Sources
Permissible Internal Dose
X-rays up to Two Million Volts
Heavy Particles (Neutrons, Protons, and Heavier)
Electrons, Gamma Rays and X-rays Above Two Million Volts
Handling of Radioactive Isotopes and Fission Products
Monitoring Methods and Instruments
Waste Disposal and Decontamination
Protection Against Radiations from Ra, Co^{60}, and Cs^{137} Encapsulated Sources
Regulation of Radiation Exposure by Legislative Means
Incineration of Radioactive Waste
Electron Protection
Safe Handling of Cadavers Containing Radioactive Isotopes
Permissible Exposures Under Emergency Conditions

In 1955 it was decided, at the suggestion of the American College of Radiology, to expand the activities of the National Committee to include consideration of problems of radiation dosimetry and measurement, and for this purpose the following subcommittees were added:

M-1. Standards and Measurement of Radioactivity for Radiological Use
M-2. Standards and Measurement of Radiological Exposure Dose
M-3. Standards and Measurement of Absorbed Radiation Dose
M-4. Relative Biological Effectiveness

Since its inception, the National Committee has provided for the protection of those occupationally exposed to radiations, and of the public, by setting the rules for building protection in areas where ionizing radiations are used and for the protection to be built into equipment. The principles established by the Committee were adopted by the Metallurgical Project (Atom Bomb) and resulted in a far safer industrial operation, so far as radiation damage was concerned, than has existed in any industry with regard to any other hazard.

The Committee's recommendations have never been legally binding on any one. Taylor, in discussing this phase in 1932, said: "The question of the legal status of these recommendations has been frequently raised. They have none. The committee feels that none is needed; that legislative enactment tends to stunt development and prevent healthy changes. We are free to admit that our present proposals may require changes in the future as they are developed. We wish nothing to interfere with the freedom for modification." Several times since then the recommendations have been changed, the latest modifications appearing in RADIOLOGY in February 1957.

The philosophy of most radiologists throughout the past sixty years has been to expose themselves, their patients, and their employees to the least possible radiation consistent with the best interest of the patient. The Committee has frequently recommended "that exposure to radiation be kept at the lowest practicable level in all cases."

The problem of exposure to radiation is now passing into the hands of many people who have no training or experience in the biological effects. For them, more definite and stricter rules need to be—and are being—established, but radiologists will always strive to keep well below any maximum permissible levels.

Recognizing all the past history, the Radiological Society of North America, at its Forty-second Annual Meeting, in December 1956, unanimously passed the following resolution:

"The Radiological Society of North America desires to call attention to the following facts:

"*First*, that organized radiologists formed a committee of the American Roentgen Ray Society to study methods of protection against ionizing radiation in 1920.

"*Second*, that these studies were continued and expanded by the Advisory Committee on X-Ray and Radium Protection formed in 1929 on a national

cooperative basis under the sponsorship of the National Bureau of Standards.

"*Third*, that, following World War II and the birth of the Age of Nuclear Power, the Advisory Committee was expanded into the National Committee on Radiation Protection, taking on representatives of all the new agencies concerned with ionizing radiation, including in its membership geneticists.

"*Fourth*, that those committees, cooperating with the International Commission on Radiologic Protection, have been studying the biologic effects of radiations and how to protect radiation workers and the public from such effects during all of these years.

"In view of these facts, it is not possible for us to *initiate* a movement for protection as requested by the National Academy of Sciences–National Research Council, but the *Radiological Society of North America* hereby resolves to *continue* its vigorous efforts to reduce the radiation exposure from all ionizing radiations to the lowest limit consistent with medical necessity, and in particular to make sure that proper safeguards are taken to minimize the radiation dose to the reproductive cells."

ROBERT S. STONE, M. D.

ANNOUNCEMENTS AND BOOK REVIEWS

AMERICAN BOARD OF RADIOLOGY

At a recent meeting, held in Tampa, Florida, the American Board of Radiology elected the following officers for the coming year: Dr. Donald S. Childs, Syracuse, N. Y., President; Dr. Bernard P. Widmann, Philadelphia, Penna., Vice-President; Dr. H. Dabney Kerr, St. Michaels, Md., Secretary; Dr. James W. J. Carpender, Chicago, Ill., Treasurer.

FILMS WANTED

Interesting cases are sought for the Sunday evening Film Reading session at the November meeting of the Radiological Society of North America. Cases will be selected primarily for their educational value. Those wishing to participate, are requested to submit, along with the radiographs, full clinical and pertinent laboratory data. A diagnosis must be possible in each case either by radiographic examination alone or through a combination of radiographic, clinical, and laboratory information. All cases must have reasonable proof of the diagnosis, preferably anatomical confirmation.

Cases should be forwarded to the moderator, Dr. John A. Evans, The New York Hospital-Cornell Medical Center, 525 East 68th St., New York 21, N. Y. The material will be returned to the participants following completion of the program.

An early response to this invitation is urged. No cases will be accepted after Nov. 1.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS

Dr. Paul Riemenschneider, Department of Radiology, Medical College, State University of New York, Syracuse, N. Y., has been elected the new Secretary-Treasurer of the Association of University Radiologists. Other officers are: Dr. Roger A. Harvey, Chicago, Ill., President, and Dr. David M. Gould, Little Rock, Ark., President-Elect.

ARKANSAS RADIOLOGICAL SOCIETY

The Arkansas Radiological Society recently elected the following officers: President, B. A. Rhinehart, M.D., Little Rock; Vice-President, Edwin Gray, M.D., Little Rock; Secretary-Treasurer, E. A. Mendelsohn, M.D., 1500 Dodson Ave., Fort Smith.

CALIFORNIA MEDICAL ASSOCIATION, SECTION ON RADIOLOGY

At a recent meeting of the California Medical Association, Section on Radiology, the following officers were chosen for the ensuing year: Chairman, Stanford B. Rossiter, M.D., of Menlo Park; Secretary, Nathan M. Spishakoff, M.D., 405 North Bedford Drive, Beverly Hills; Assistant Secretary, William H. Graham, M.D., 630 E. Santa Clara St., San Jose.

CHICAGO ROENTGEN SOCIETY

The newly elected officers of the Chicago Roentgen Society are: Dr. Ervin F. Hummon, Jr., President; Dr. Edward George Warnick, Vice-President; Dr. Arthur S. J. Petersen, 11406 S. Parnell Ave., Chicago 28, Secretary-Treasurer.

INDIANA ROENTGEN SOCIETY

The Indiana Roentgen Society has elected the following officers for the coming year: President, Arthur A. Hobbs, M.D., of Evansville; Vice-President, Wallace D. Buchanan, M.D., of South Bend; Secretary-Treasurer, Chester A. Stayton, Jr., M.D., 313 Hume Mansur Building, Indianapolis 4.

MEMPHIS ROENTGEN SOCIETY

At a recent meeting, the following were elected to office in the Memphis Roentgen Society: President, Dr. David S. Carroll; Vice-President, Dr. Dominic J. Cara; Secretary-Treasurer, Dr. James L. Booth, 899 Madison Ave., Memphis; Counselor to the American College of Radiology, Dr. J. E. Whiteleather, and Alternate Counselor, Dr. John M. Wilson.

RADIOLOGICAL SOCIETY OF NEW JERSEY

At the annual meeting of the Radiological Society of New Jersey, in May, the following officers were elected for 1957–58: President, Dr. Louis Jack Levinson of Newark; Vice-President, Dr. George G. Green of Asbury Park; Secretary, Dr. Andrew P. Dedick, Jr., 67 E. Front St., Red Bank; Treasurer, Dr. William L. Palazzo, of Teaneck.

MIDSUMMER CONFERENCE ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

The Nineteenth Midsummer Conference of the Rocky Mountain Radiological Society will be held Aug. 15–17 in the Shirley-Savoy Hotel, Denver, Colo. The guest speakers will be Benjamin H. Orndoff, M.D., Chicago; Paul C. Aebersold, Ph.D., Oak Ridge; Titus C. Evans, Ph.D., Iowa City; Harry M. Spence, M.D., Dallas; Russell H. Morgan, M.D., Baltimore; and Henry L. Jaffe, M.D., Los Angeles. Others participating in the program will be Dr. John S. Bouslog, Denver; Dr. C. Edgar Virden,

Kansas City, Mo.; Dr. George R. Buck, Denver; Dr. Irvin E. Hendryson, Denver; Dr. Gerald S. Maresh, Denver; Dr. Ira H. Lockwood, Kansas City, Mo.; Drs. Kenneth D. A. Allen, Robert W. Lackey, and Garret B. Byma, Denver; Dr. Grant P. Raitt, Billings, Mont; Dr. Charles T. Dotter, Portland, Ore.; Drs. R. E. Collier and A. D. Sears, Dallas; Drs. J. Gershon-Cohen, Victor Kremens, and Simon M. Berger, Philadelphia; Drs. Hillier L. Baker, Jr., and John R. Hodgson, Rochester, Minn.; Drs. John T. Mallams and J. E. Miller, Dallas; Drs. Leo S. Figiel and Steven J. Figiel, Detroit; Dr. Albert G. Barsh, Lubbock, Texas; Dr. Benjamin Milder, St. Louis; Dr. Sidney W. Nelson, Columbus, Ohio; Dr. Peter E. Russo, Oklahoma City; and Dr. William R. Christensen, Dr. Ralph R. Meyer, and Dr. Angus K. Wilson, Salt Lake City.

Special features are the joint meeting with the Denver Medical Society, Thursday evening, Aug. 15, preceded by an informal Guest Speakers' Dinner; the annual banquet Friday evening; and a Saturday trip to Central City where, following dinner at the Teller House, there will be an opportunity to attend a play at the Central City Opera House.

SOUTH CAROLINA RADIOLOGICAL SOCIETY

At the May meeting of the South Carolina Radiological Society, the following men were elected to office for the coming year; Dr. William Klauber of Greenwood, President; Dr. Sam Fisher of Greenville, Vice-President; Dr. Wayne Reeser, 1600 Ninth Ave., Conway, Secretary-Treasurer.

TRI-STATE RADIOLOGICAL SOCIETY

The Tri-State Radiological Society elected the following new officers at a recent meeting: President, A. A. Sullenger, M.D., of Vincennes, Ind.; President-Elect, E. L. Hendershot, M.D., of Evansville, Ind.; and Secretary-Treasurer, Robert E. Beck, M.D., 600 Mary St., Evansville, Ind.

INTER-AMERICAN CONGRESS OF RADIOLOGY

The Sixth Inter-American Congress of Radiology will be held in Lima, Peru, Nov. 2–8, 1958. All inquiries concerning the Congress should be addressed to the Secretary for North America, Dr. Jorge de la Flor, Hospital Arzobispo Loayzo, Lima, Peru.

EXHIBITION OF X-RAY APPARATUS

In conjunction with the Annual Congress of the British Institute of Radiology, the British x-ray industry is holding an exhibition of medical x-ray apparatus at the Royal Horticultural Society's Hall, Westminster, London, Nov. 27–29, 1957.

Tickets are available on request from The Secretary, British Institute of Radiology, 32 Welbeck St., London W. 1, England.

Books Received

Books received are acknowledged under this heading, and such notice may be regarded as recognition of the courtesy of the sender. Reviews will be published in the interest of our readers and as space permits.

INTERNATIONAL CONGRESS OF GASTROENTEROLOGY. FIFTH MEETING OF L'ASSOCIATION DES SOCIÉTÉS EUROPÉENNES ET MÉDITERRANÉENNES DE GASTRO-ENTÉROLOGIE, LONDON, JULY 18–21, 1956. President of the Congress: Dr. Thomas Hunt. (Reprint from *Gastroenterologia*, Vol. 86, Nos. 3–5, 1956.) A volume of 766 pages, with illustrations. Published by S. Karger AG., Arnold Böcklinstrasse 25, Basel, Switzerland. Distributed in the United States by Albert J. Phiebig, P. O. B. 352, White Plains, N. Y., 1957.

Book Reviews

FUNDAMENTALS OF CLINICAL FLUOROSCOPY, WITH ESSENTIALS OF ROENTGEN INTERPRETATION. By CHARLES B. STORCH, M.D., Associate Attending Roentgenologist, Radiodiagnostic Department, Beth-El Hospital, Brooklyn, New York. A volume of 306 pages, with 318 illustrations. Published by Grune & Stratton, Inc., New York. Second Revised Edition, 1957. Price $8.75.

This is the second edition of a text on clinical fluoroscopy first published in 1951, with the inclusion of more recent methods and subjects not previously covered. New sections on the study of congenital heart disease and of the gastrointestinal tract have been added.

The text is designed to serve as a practical approach to fluoroscopy as an everyday procedure in clinical practice, with due consideration of its possibilities and limitations. The first chapter outlines the basic concepts, giving attention to well known dangers and the need and methods of protection. Image intensification is also described. Subsequent chapters detail the fluoroscopic study of the various organs of the body.

A valuable bibliography is included and there is a short but adequate index. The book will be of special value to residents and others wishing to learn the basic technic of fluoroscopy. To experienced radiologists it will serve as a useful reference source. Not the least of its appeal to the former group will lie in the attention given to various stumbling blocks encountered by the beginner.

NOTES ON ATOMIC ENERGY FOR MEDICAL OFFICERS: AN INTRODUCTION TO THE SUBJECT FOR SERVICE AND OTHER MEDICAL OFFICERS WHO MAY BE CONCERNED WITH DEFENCE AGAINST ATOMIC BOMBS AND SIMILAR PROBLEMS. Prepared by the Staff of the Royal Naval Medical School, Alverstoke, England. A volume of 170 pages, with 12 plates. Published by Harrison & Sons, London; Philosophical Library, New York, N. Y. Price $4.75.

This is a schoolmaster attempt to cover everything in small space without trusting the reader to know very much to begin with. It was apparently written in 1950, although a four-page appendix on the thermonuclear weapon speaks of the 1954 test.

The first fifty pages (six of the sixteen chapters) are devoted to a review of physics before an explanation is attempted of atomic energy and the bomb and its physical effects. Genetics and the effects of radiation on cells and on the body are considered in thirty-six pages. The chapters on military hazards and treatment of casualties are extremely brief. Monitoring instruments are described in minimum detail, and necessary facts are given about protection against radiation. Appendixes supply pertinent physical constants of atoms and energy in excellently chosen brevity, maximum permissible radiation hazard (not down to date) with a brief discussion of genetic dangers, and the briefest review of uses of isotopes.

In a good many places the presentation is well designed to give unprepared minds a conception about which it is not easy to be precise. Elsewhere statements are incomplete or questionable, due either to too great compromise with space limitations or the writers' lack of a sufficiently deep knowledge of the very broad field they have undertaken to cover. An example is the paragraph on x-ray tubes, hot cathode *vs.* gas tubes. It may be quibbling to complain of the statement that x-rays are never refracted and, again, that all the radiations from radioactive elements come from their nuclei. A serious misstatement, however, is the doubling dose for mutation rate, given as 50 r for *Drosophila*. The importance of the intestine in the lethality of middling doses of radiation is not set forth.

In the chapter on protection, the half-value layers for 3-MEV gammas are exaggerated by about one-half—a greater thickness than is given in *Handbook 55* for 7.5-MEV photons.

British spellings and abbreviations are an occasional annoyance to an American reader, but the only serious error in taste is the placing of the atomic number as a lower right subscript to the atomic symbol, a place properly reserved for the number of those atoms in the molecule.

References are given with each chapter, and there is at the end a bibliography of popular and more advanced books. Line drawings are numbered by chapters, and twelve half-tone plates are inserted just before the rather meager index.

An able instructor might find this work a good skeleton upon which to hang an indoctrination course.

PROCEEDINGS OF THE THIRD NATIONAL CANCER CONFERENCE. Detroit, Michigan, June 4–6, 1956. Sponsored by American Cancer Society, Inc., and National Cancer Institute, U.S. Public Health Service. A volume of 962 pages, with numerous illustrations and tables. Published by J. B. Lippincott Co., Philadelphia, 1957. Price $9.00.

In this volume are brought together the papers presented at the Third National Cancer Conference held in Detroit in June 1956 under the sponsorship of the American Cancer Society, the National Cancer Institute, and the U. S. Public Health Service

Part I consists of the opening and closing remarks; Part II reports six general lectures; Part III comprises the bulk of the scientific material which was presented in the form of Symposia and Panel Discussions on specific problems of major importance. The complete coverage of these subjects is exemplified by the symposium on cancer of the breast with thirteen contributors plus five discussants.

The participants in the conference were scientists of first rank, giving added authority to the contents of this volume. It is recommended to all those interested in the diagnosis and treatment of malignant diseases.

THE DOCTOR AS A WITNESS. By JOHN EVARTS TRACY, Professor of Law (Emeritus), University of Michigan, Ann Arbor, Mich. A volume of 222 pages. Published by W. B. Saunders Co., Philadelphia and London, 1957. Price $4.25.

This small volume contains a great deal of information which cannot fail to be of benefit to any doctor who is frequently, or even occasionally, called to serve as a witness in a court of law. The eminence of the author lends much weight to the opinions expressed.

The privileges and obligations of the doctor-witness are considered and a clear discussion is given of opinion evidence and expert testimony. Chapters are devoted to direct and cross-examination, testimony on the issue of insanity, workmen's compensation, and malpractice cases. Others concern preparation for trial, what makes a good medical witness, and the compensation of the doctor-witness.

The text is simply written without an excess of legal terminology. The book is highly recommended as fulfilling the author's stated purpose, "to explain to the busy practitioner who has had little experience in court rooms the various kinds of legal proceedings in which he may be called to testify, how these proceedings are conducted, and what is expected from a medical witness when he takes the stand."

STRAHLENDOSIS UND STRAHLENWIRKUNG. TAFELN UND ERLÄUTERUNGEN; UNTERLAGEN FÜR DEN STRAHLENSCHUTZ. By B. RAJEWSKY, with the collaboration of K. AURAND, O. HUG, H. MERGLER, H. MUTH, H. PAULY, A. SCHRAUB, E. SIX, AND I. WOLF, Max-Planck-Institut für Biophysik, Frankfurt a. M. A volume of 486 pages, including numerous graphs and tables. Published by Georg Thieme, Stuttgart, 2d ed., 1956. Distributed in the United States and Canada by the Intercontinental Medical Book Corp., New York 16, N. Y. Price DM 36.—($8.60)

The first edition of this remarkable collection of information on various types of radiation and on the injurious and other effects produced by them was reviewed in RADIOLOGY about two and a half years ago (**64**:278, 1955). That edition was out of print in four months and the editor and publisher wisely decided not to reprint, but instead to bring out a second edition. The result is a volume in which the text has been increased by 17 per cent, the graphs and tables by 29 per cent, the reference list by 229 per cent, and the price by 327 per cent. The book is again divided into three parts: A. Introduction and Explanation of the Tables (pages numbered 1 to 72); B. Tables (pages unnumbered but marked with code letters referring to the various chapters in section A); C. Bibliography (pages numbered 1 to 256). Parts A and B deal with such subjects as the various types of radiation, acute radiation sickness, whole body irradiation, fractionation and protraction, maximum permissible dosages, radiation reactions of various organs and blood, radiation cancer, relative biological effectiveness of ionizing radiations, permissible doses of radioisotopes, and atom bomb explosion effects. Several new tables have been added and others brought up to date. The third part, the bibliography, presents the largest addition to the book. It comprises 256 pages or more than one-half of the total number of pages. The list of 3,100 references which appeared in the first edition is reprinted and a second separate list of about the same number of references has been added, covering the literature more or less adequately up to the end of 1955.

A new feature of the second edition is an index of twenty pages, which, however, due to the peculiar organization of the various sections of the book, seems to be more confusing than helpful. This enlarged collection of data on radiation dosages and radiation effects can again be highly recommended and should be in the library of everyone who deals with ionizing radiations.

ACTIONS CHIMIQUES ET BIOLOGIQUES DES RADIATIONS. Collection dirigée par M. HAISSINSKY. Deuxième série. I. Les effets chimiques produits par les rayons ionisants en phase gazeuse, by W. MUND. II. Phénomènes de luminescence provoqués par les rayonnements de grande énergie, by M. AGENO. III. Introduction à la dosimétrie des radiations, by N. MILLER. A volume of 222 pages, with 39 figures. Published by Masson et Cie, 120, Boulevard Saint-Germain, Paris 6e, 1956. Price 2,800 fr. (paper bound), 3,400 fr. (cloth).

This monograph comprises three essays on the chemical and biologic action of radiations. An earlier series, published in 1955 under the same title, included papers on The Physical Aspects of Radiobiology, by L. H. Gray; Chemistry of Radiations of Aqueous Solutions, by M. Lefort; Modern Trends in Radiation Biochemistry, by W. M. Dale. In this second series we have The Chemical Effects Produced by Ionizing Rays in the Gaseous Phase, by W. Mund; Phenomena of Luminescence Provoked by Radiations of Great Energy, by M. Ageno; Introduction to Dosimetry of Radiations, by N. Miller.

Mund gives a general preliminary discussion on possible modes of direct and indirect actions of radiation in the gaseous phase and from this proceeds to discuss the effects in specific inorganic and organic gaseous media.

Ageno discusses the phenomena of luminescence in a general way, including historical aspects, methods of observing and measuring scintillations, the production of luminescence in crystals, liquids and solids, and the transport and mechanism of scintillations.

Miller begins his essay on dosimetry by a discussion of the dissipation of energy of ionizing radiations as they traverse various states of matter. Methods of measuring energy, such as calorimetry, collection of charges, nuclear reactions in solutions, ionization chambers, and special considerations of dosimetry in the gaseous phase are all included.

For anyone with a good reading knowledge of French and a fairly good background of knowledge in biophysics there is considerable material for reference, especially in relation to biophysical research, in the material presented.

RADIOLOGICAL SOCIETIES: SECRETARIES AND MEETING DATES

Editor's Note: Secretaries of state and local radiological societies are requested to co-operate in keeping this section up-to-date by notifying the editor promptly of changes in officers and meeting dates.

RADIOLOGICAL SOCIETY OF NORTH AMERICA. *Secretary-Treasurer*, Donald S. Childs, M.D., 713 E. Genesee St., Syracuse 2, N. Y.

AMERICAN RADIUM SOCIETY, *Secretary*, Theodore R. Miller, M.D., 139 E. 36 St., New York 16, N. Y.

AMERICAN ROENTGEN RAY SOCIETY. *Secretary*, Barton R. Young, M.D., Germantown Hospital, Philadelphia 44, Penna.

AMERICAN COLLEGE OF RADIOLOGY. *Exec. Secretary*, William C. Stronach, 20 N. Wacker Dr., Chicago 6.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS. *Secretary-Treasurer*, Paul Riemenschneider, M.D., Department of Radiology, State University of New York, Syracuse, N. Y.

SECTION ON RADIOLOGY, A. M. A. *Secretary*, T. Leucutia, M.D., 10 Peterboro, Detroit 1, Mich.

SOCIETY OF NUCLEAR MEDICINE. *Secretary*, Robert W. Lackey, M.D., 452 Metropolitan Bldg., Denver 2, Colo.

Alabama

ALABAMA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, J. A. Meadows, Jr., M.D., Medical Arts Bldg., Birmingham 5.

Arizona

ARIZONA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, R. Lee Foster, M.D., 1313 N. Second St., Phoenix. Annual meeting with State Medical Association; interim meeting in December.

Arkansas

ARKANSAS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, E. A. Mendelsohn, M.D., Holt-Krock Clinic, Fort Smith. Meets quarterly.

California

CALIFORNIA MEDICAL ASSOCIATION, SECTION ON RADIOLOGY. *Secretary*, Nathan M. Spishakoff, M.D., 405 N. Bedford Drive, Beverly Hills.

EAST BAY ROENTGEN SOCIETY. *Secretary*, Dan Tucker, M.D., 434 30th St., Oakland 9. Meets monthly, first Thursday, at Peralta Hospital.

LOS ANGELES RADIOLOGICAL SOCIETY. *Secretary*, Lewis J. Peha, M.D., 405 N. Bedford Dr., Beverly Hills. Meets monthly, second Wednesday, Los Angeles County Medical Association Bldg.

NORTHERN CALIFORNIA RADIOLOGICAL CLUB. *Secretary*, H. B. Steward, Jr., M.D., 2920 Capitol Ave., Sacramento. Meets last Monday of each month, September to May.

PACIFIC ROENTGEN SOCIETY. *Secretary*, L. Henry Garland, M.D., 450 Sutter St., San Francisco 8. Meets annually at time of California State Medical Association convention.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA. *Secretary-Treasurer*, Harold P. Tompkins, M.D., 658 S. Westlake, Los Angeles 57.

SAN DIEGO RADIOLOGICAL SOCIETY. *Secretary*, C. W. Bruner, M.D., 2456 Fourth Ave., San Diego 1. Meets first Wednesday of each month.

SAN FRANCISCO RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Irma Smith, M.D., 450 Sutter St., San Francisco 8. Meets quarterly, at Grison's Steak House.

SOUTH BAY RADIOLOGICAL SOCIETY. *Secretary*, Howard L. Jones, M.D., Palo Alto Hospital, Palo Alto. Meets monthly, second Wednesday.

X-RAY STUDY CLUB OF SAN FRANCISCO. *Secretary*, John H. Heald, M.D., 450 Sutter St., San Francisco 8. Meets third Thursday at 7.30, Children's Hospital, September through June.

Colorado

COLORADO RADIOLOGICAL SOCIETY. *Secretary*, Lorenz R. Wurtzebach, M.D., 601 E. Nineteenth Ave., Denver 5. Meets monthly, third Friday, at University of Colorado Medical Center or Denver Athletic Club.

Connecticut

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY. *Secretary-Treasurer*, Ralph J. Littwin, M.D., Bristol Hospital, Bristol. Meets bimonthly, second Wednesday.

District of Columbia

RADIOLOGICAL SECTION DISTRICT OF COLUMBIA MEDICAL SOCIETY. *Secretary-Treasurer*, Melvin O. Goodman, M.D., 1930 K St., N.W., Washington 6. Meets third Wednesday, January, March, May, and October, 8:00 P.M., in Medical Society Library.

Florida

FLORIDA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, C. Robert DeArmas, M.D., 135 Broadway, Daytona Beach. Meets in April and in October.

GREATER MIAMI RADIOLOGICAL SOCIETY, *Secretary-Treasurer*, George P. Daurelle, M.D., Jackson Memorial Hospital, Miami 36. Meets monthly, third Wednesday, 8:00 P.M., at Mercy Hospital.

NORTH FLORIDA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Marvin Harlan Johnston, M.D., Five Points Medical Center, Jacksonville 4. Meets quarterly, March, June, September, and December.

Georgia

ATLANTA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Bert H. Malone, M.D., 1406 Reynolds St., Brunswick. Meets second Friday, September to May.

GEORGIA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Herbert M. Olnick, M.D., 417 Persons Bldg., Macon, Ga. Meets in November and at the annual meeting of the State Medical Association.

RICHMOND COUNTY RADIOLOGICAL SOCIETY. *Secretary*, Wm. F. Hamilton, Jr., M.D., University Hospital, Augusta. Meets first Thursday of each month.

Hawaii

RADIOLOGICAL SOCIETY OF HAWAII. *Secretary-Treasurer*, Richard D. Moore, M.D., St. Francis Hospital, Honolulu 17. Meets third Monday of each month.

Illinois

CHICAGO ROENTGEN SOCIETY. *Secretary-Treasurer*, Arthur S. J. Petersen, M.D., 11406 Parnell Ave., Chicago 28. Meets at the Sheraton Hotel, second Thursday of October, November, January, February, March, and April at 8:00 P.M.

ILLINOIS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Stephen L. Casper, M.D., Physicians and Surgeons Clinic, Quincy.

ILLINOIS STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY. *Secretary*, George E. Irwin, Jr., M.D., 427 N. Main St., Bloomington.

Indiana

INDIANA ROENTGEN SOCIETY. *Secretary-Treasurer*, Chester A. Stayton, Jr., M.D., 313 Hume-Mansur Bldg., Indianapolis 4. Meets twice a year, first Sunday in May and during fall meeting of State Medical Association.

TRI-STATE RADIOLOGICAL SOCIETY (Southern Indiana, Northwestern Kentucky, Southeastern Illinois). *Secretary-Treasurer*, Robert E. Beck, M.D., 600 Mary St., Evansville, Ind. Meets last Wednesday, October, January, March, and May, 8:00 P.M., at the Elks' Club, Evansville, Ind.

Iowa

IOWA RADIOLOGICAL SOCIETY. *Secretary*, James T. McMillan, M.D., 1104 Bankers Trust Bldg., Des Moines. Meets during annual session of State Medical Society, and in the Fall.

Kansas

KANSAS RADIOLOGICAL SOCIETY. *Secretary*, James R. Stark, M.D., 3244 East Douglas St., Wichita. Meets in the Spring with the State Medical Society and in the Winter on call.

Kentucky

KENTUCKY RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Robert H. Akers, M.D., 1405 West Broadway, Louisville 3. Meets monthly, second Friday, at Seelbach Hotel, Louisville.

Louisiana

ORLEANS PARISH RADIOLOGICAL SOCIETY. *Secretary*, Joseph V. Schlosser, M.D., Charity Hospital of Louisiana, New Orleans 13. Meets second Tuesday of each month.

RADIOLOGICAL SOCIETY OF LOUISIANA. *Secretary-Treasurer*, W. S. Neal, M.D., 602 Pere Marquette Bldg., New Orleans.

SHREVEPORT RADIOLOGICAL CLUB. *Secretary*, W. R. Harwell, M.D., 608 Travis St. Meets monthly September to May, third Wednesday.

Maine

MAINE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Francis J O'Connor, M.D., Augusta General Hospital, Augusta. Meets in June, October, December, and April.

Maryland

BALTIMORE CITY MEDICAL SOCIETY, RADIOLOGICAL SECTION. *Secretary-Treasurer*, James K. V. Willsod, M.D., 1100 N. Charles St., Baltimore 1. Meets third Tuesday, September to May.

MARYLAND RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Nathan B. Hyman, M.D., 1805 Eutaw Place, Baltimore 17.

Michigan

DETROIT X-RAY AND RADIUM SOCIETY. *Secretary-Treasurer*, Joseph O. Reed Jr., M.D., 3825 Brush St., Detroit 1. Meets first Thursday, October to May, at Wayne County Medical Society club rooms.

UPPER PENINSULA RADIOLOGICAL SOCIETY. *Secretary*, Arthur Gonty, M.D., Menominee. Meets quarterly.

Minnesota

MINNESOTA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, O. J. Baggenstoss, M.D., 1953 Medical Arts Bldg., Minneapolis 2. Meets three times a year, in Fall, Winter, and Spring.

Mississippi

MISSISSIPPI RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Robert P. Henderson, M.D., 316 Medical Arts Bldg., Jackson. Meets monthly, on third Tuesday, at 6:30 P.M., at the Hotel Edwards, Jackson.

Missouri

RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY. *Secretary-Treasurer*, D. R. Germann, M.D., University of Kansas Medical Center, Kansas City 3, Kans. Meets last Friday of each month.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS. *Secretary*, Thomas F. Maher, M.D., 634 N. Grand Blvd., St. Louis, Mo. Meets on fourth Wednesday, October to May.

Montana

MONTANA RADIOLOGICAL SOCIETY. *Secretary*, John Stewart, M.D., Billings Clinic, Billings. Meets annually.

Nebraska

NEBRASKA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, James F. Kelly, Jr., M.D., 816 Medical Arts Bldg., Omaha. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England

CONNECTICUT VALLEY RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Thomas J. Crowe, M.D., 53 Center St., Northampton, Mass. Meets second Friday of October and April.

NEW ENGLAND ROENTGEN RAY SOCIETY. *Secretary*, Raymond A. Dillon, M.D., 24 Wedgemere Ave., Winchester, Mass. Meets monthly on third Friday, October through May, at the Hotel Commander, Cambridge, Mass.

New Hampshire

NEW HAMPSHIRE ROENTGEN SOCIETY. *Secretary*, Albert C. Johnson, M.D., 127 Washington St., Keene.

New Jersey

RADIOLOGICAL SOCIETY OF NEW JERSEY. *Secretary*, Andrew D. Dedick, Jr., M.D., 67 E. Front St., Red Bank. Meets at Atlantic City at time of State Medical Society and midwinter in Elizabeth.

New York

BROOKLYN RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Harold N. Schwinger, M.D., 62 Eighth Ave. Meets first Thursday, October through May.

BUFFALO RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Charles Bernstein, M.D., 685 Delaware Ave., Buffalo. Meets second Monday, October to May.

CENTRAL NEW YORK ROENTGEN SOCIETY. *Secretary*, Dwight V. Needham, M.D., 608 E. Genesee St., Syracuse 2. Meets in January, May, and October.

KINGS COUNTY RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Ernest I. Melton, M.D., 2187 Ocean Ave., Brooklyn. Meets fourth Thursday, October to April (except December), at 9:00 P.M., Kings County Medical Bldg.

NASSAU RADIOLOGICAL SOCIETY. *Secretary*, Jerome Zwanger, M.D., 126 Hicksville Road, Massapequa. Meets second Tuesday, February, April, June, October, and December.

NEW YORK ROENTGEN SOCIETY. *Secretary*, Harold G. Jacobson, M.D., Montefiore Hospital, 210th St. and Bainbridge Ave., New York 67, N. Y.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Irving Van Woert, Jr., M.D., Albany Hospital, Albany. Meets in the capital area second Wednesday, October, November, March, and April. Annual meeting in May or June.

RADIOLOGICAL SOCIETY OF NEW YORK STATE. *Secretary-Treasurer*, Mario C. Gian, M.D., 610 Niagara St., Buffalo 1. Meets annually with the State Medical Society.

ROCHESTER ROENTGEN-RAY SOCIETY. *Secretary-Treasurer*, John W. Colgan, M.D., 277 Alexander St., Rochester 18. Meets at Strong Memorial Hospital, 8:15 P.M., last Monday of each month, September through May.

WESTCHESTER RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Arnold Myron Wald, M.D., 406 Boston Post Road, Port Chester. Meets third Tuesday of January and October and at other times as announced.

North Carolina

RADIOLOGICAL SOCIETY OF NORTH CAROLINA. *Secretary*, William H. Sprunt, M.D., North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in April and October.

North Dakota

NORTH DAKOTA RADIOLOGICAL SOCIETY. *Secretary*, Marianne Wallis, M.D., Minot. Meets in the Spring with State Medical Association; in Fall or Winter on call.

Ohio

OHIO STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John R. Hannan, M.D., 10515 Carnegie Ave., Cleveland 6.

CENTRAL OHIO RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Arthur R. Cohen, M.D., 41 S. Grant Ave., Columbus. Meets second Thursday, October, November, January, March, and May, 6:30 P.M., Fort Hayes Hotel, Columbus.

CLEVELAND RADIOLOGICAL SOCIETY. *Secretary*, Frederick A. Rose, M.D., 2065 Adelbert Road, Cleveland 6. Meets at 7:00 P.M., fourth Monday, October, November, January, February, March and April, at Tudor Arms Hotel.

GREATER CINCINNATI RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Morris M. Garrett, M.D., 630 Scott Str., Covington, Ky. Meets first Monday, September to June, at Cincinnati General Hospital.

MIAMI VALLEY RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, H. D. Robertson, M.D., Miami Valley Hospital, Dayton 9. Meets monthly, second Friday.

Oklahoma

OKLAHOMA STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Sol Wilner, M.D., Medical Arts Bldg., Tulsa.

Oregon

OREGON RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, N. L. Bline, M.D., 806 S.W. Broadway, Portland 5. Meets monthly, second Wednesday, October to June, at 8:00 P.M., University Club, Portland.

Pacific Northwest

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY. *Secretary-Treasurer*. Robert Hanf, M.D., 807 South Auburn, Kennewick, Wash. Meets annually in May.

Pennsylvania

PENNSYLVANIA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Walter P. Bitner, M.D., 234 State St., Harrisburg. Meets annually.

PHILADELPHIA ROENTGEN RAY SOCIETY. *Secretary*, Roderick L. Tondreau, M.D., 3400 Spruce St., Philadelphia 4. Meets first Thursday of each

month at 5:00 P.M., from October to May, in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY. *Secretary*, Edward M. Schultz, M.D., 3401 Fifth Ave., Pittsburgh 13. Meets monthly, second Wednesday, at 6:30 P.M., October to May, at the Hotel Roosevelt.

Rocky Mountain States

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John H. Freed, M.D., 4200 E. Ninth Ave., Denver 7, Colo.

South Carolina

SOUTH CAROLINA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Wayne Reeser, M.D., 1600 Ninth Ave., Conway. Meets with State Medical Association in May.

South Dakota

RADIOLOGICAL SOCIETY OF SOUTH DAKOTA. *Secretary-Treasurer*, Donald J. Peik, M.D., 303 S. Minnesota Ave., Sioux Falls. Meets during annual meeting of State Medical Society.

The Southeast

Southern Radiological Conference. *Secretary-Treasurer*, Marshall Eskridge, M.D., 1252 Springhill Ave., Mobile, Ala.

The Southwest

SOUTHWESTERN RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Gordon L. Black, M.D., 1501 Arizona Bldg., El Paso.

Tennessee

MEMPHIS ROENTGEN SOCIETY. *Secretary-Treasurer*, James L. Booth, M.D., 899 Madison Ave., Memphis 3. Meets monthly first Monday, John Gaston Hospital.

TENNESSEE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, George K. Henshall, M.D., 311 Medical Arts Bldg., Chattanooga 3. Meets annually with State Medical Association in April.

Texas

DALLAS-FORT WORTH RADIOLOGICAL CLUB, *Secretary*, Albert H. Keene, M.D., 3707 Gaston Ave., Suite 116, Dallas. Meets monthly, third Monday, 6:30 P.M., at the Greater Fort Worth International Airport.

HOUSTON RADIOLOGICAL SOCIETY. *Secretary*, John M. Phillips, M.D., Hermann Hospital, Houston 25. Meets fourth Monday at the Doctors' Club.

SAN ANTONIO-MILITARY RADIOLOGICAL SOCIETY. *Secretary*, Hugo F. Elmendorf, Jr., M.D., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets at Brook Army Medical Center, second Wednesday of each month.

TEXAS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Jarrell E. Miller, M.D., 3500 Gaston Ave., Dallas 4.

Utah

UTAH STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Angus K. Wilson, M.D., 343 S. Main St., Salt Lake City 1. Meets third Wednesday, January, March, May, September, November.

Virginia

VIRGINIA RADIOLOGICAL SOCIETY. *Secretary*, P. B. Parsons, M.D., 1308 Manteo St., Norfolk 7.

Washington

WASHINGTON STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Eva L. Gilbertson, M.D., 1317 Marion St., Seattle 4. Meets fourth Monday, September through May, at 610 Pine St., Seattle.

West Virginia

WEST VIRGINIA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, W. Paul Elkin, M.D., 515–519, Medical Arts Bldg., Charleston. Meets concurrently with annual meeting of State Medical Society, and at other times as arranged by Program Committee.

Wisconsin

MILWAUKEE ROENTGEN RAY SOCIETY. *Secretary-Treasurer*, Jerome L. Marks, M.D., 161 W. Wisconsin Ave., Milwaukee 1. Meets monthly on fourth Monday at the University Club.

SECTION ON RADIOLOGY, STATE MEDICAL SOCIETY OF WISCONSIN. *Secretary*, Abraham Melamed, M.D., 425 E. Wisconsin Ave., Milwaukee 2. Meets in October with State Medical Society.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE. Meets first and third Thursday at 4 P.M., September to May, Service Memorial Institute.

WISCONSIN RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Farrell F. Golden, M.D., 5221 Tonyawatha Trail, Madison 4.

Puerto Rico

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA. *Secretary-Treasurer*, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, P.R.

CANADA

CANADIAN ASSOCIATION OF RADIOLOGISTS. *Honorary Secretary-Treasurer*, Guillaume Gill, M.D.; *Associate Honorary Secretary-Treasurer*, Robert G. Fraser, M.D. *Central Office*, 1555 Summerhill Ave., Montreal 25, Quebec. Meets in January and June.

LA SOCIÉTÉ CANADIENNE-FRANÇAISE D'ELECTRO-RADIOLOGIE MÉDICALES. *General Secretary*, Louis Ivan Vallée, M.D., Hôpital Saint-Luc, 1058 rue St-Denis, Montreal 18. Meets third Saturday of each month.

L'ASSOCIATION DES RADIOLOGISTS DE LA PROVINCE DE QUEBEC. ASSOCIATION OF RADIOLOGISTS OF THE PROVINCE OF QUEBEC. *Secretary*, Isadore Sedlezky, M.D., 3755 Cote St. Catherine Road, Montreal. Meets four times a year.

CUBA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPÍA DE CUBA. *Secretary*, Dr. Rafael Gomez Zaldívar. Offices in Hospital Mercedes, Havana. Meets monthly.

MEXICO

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PANAMA

SOCIEDAD RADIOLÓGICA PANAMEÑA. *Secretary-Editor*, Luis Arrieta Sánchez, M.D., Apartado No. 86, Panama, R. de P.

ABSTRACTS OF CURRENT LITERATURE

ROENTGEN DIAGNOSIS

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ROENTGEN DIAGNOSIS

THE HEAD AND NECK

The Estimation of Skull Capacity from Roentgenologic Measurements. I. L. MacKinnon, J. A. Kennedy, and T. V. Davies. Am. J. Roentgenol. **76:** 303–310, August 1956. (Department of Anatomy, University of London King's College, Strand, London, England)

The authors discuss the estimation of skull capacity from roentgenological measurements. In their study, 52 dry adult skulls of known capacity were used and anteroposterior and lateral Bucky roentgenograms were made. Their objective was to relate the known capacity of these skulls to various easily obtainable roentgenological measurements. The internal length (L), height (H), breadth (W), and diameter from the bregma to the posterior cranial fossa (B) were measured.

The estimated average capacity of any 50 of the skulls, based on any one of the four diameters, involved a moderate error, 0 to 6.7 per cent.

It is suggested that a reasonable estimate of the capacity (c.c.) of any adult skull can be obtained from the four internal roentgenological diameters (cm.) used in this investigation by calculating $[1/2\,(L \times H \times W) + 1/2\,(L \times B \times W)] \times 0.51$. A reasonable estimate of the average capacity of 50 or more skulls (c.c.) can be obtained from one linear roentgenologic measurement—the bregma-posterior cranial fossa diameter (cm.) by calculating $\Sigma(B \times 100)/N$, where N is th number of skulls.

Two roentgenograms; 1 graph; 3 tables.

ROBERT H. LEAMING, M.D.
Memorial Center, New York

The Tympanic Cavity and Auditory Ossicles. Roentgen Findings in Health and Disease. Eugene P. Pendergrass, Philip J. Hodes, Roderick L. Tondreau, and Philip A. Marden. Am. J. Roentgenol. **76:** 327–342, August 1956. (Hospital of the University of Pennsylvania, Philadelphia, Penna.)

The authors have made a detailed study of the tympanic cavity and its ossicles in health and disease. Their discussion is accompanied by clear, concise roentgenographic reproductions.

The tympanic cavity is best seen in the basilar and axial projections known, respectively, as the Hirtz and Mayer's projections. It is difficult to outline the ossicles individually because of their small size, but as a unit they are most clearly demonstrated in the basilar projection. The bony portion of the eustachian canal is also seen to best advantage in the basilar projection. A contrast medium may be introduced in retrograde fashion through the nasopharyngeal end of the eustachian canal by catheter, or advantage may be taken of a perforated tympanic membrane in chronically draining ears.

About once in every 2,000 aural patients the external auditory canal may be hypoplastic, partially obliterated, or entirely absent. This process is bilateral in about 15 per cent of the cases. Commonly associated with atresia of the external auditory canal are homolateral anomalies of the auricle, face, and skull. When the atresia is incomplete, contrasting air often depicts the character of the anomaly in routine lateral, axial, or basilar views. Contrast media are particularly useful in such instances. When the atresia is complete, the problem is far more difficult, and stereo basilar views as well as laminagrams are extremely valuable. Auditory atresia may be acquired, as a result of trauma or infection.

Roentgenological evidence of infection is reflected in a progressively decreased radiolucency in all air-filled portions of the temporal bone. The eustachian canal early loses its spear-like radiolucency, and at the same time the tympanic cavity becomes progressively less radiolucent. These changes are seen to best advantage in the basilar projection.

Aero-otitis media is characterized by a retraction of the ear drum and a shift in the axis of the ossicular chain. There may be a loss of detail in the eustachian canal, a diffuse increased density of the mastoid, opacification of the tympanic cavity, and in rare instances fluid levels within the tympanic cavity.

Forty roentgenograms; 1 diagram.

ROBERT H. LEAMING, M.D.
Memorial Center, New York

Ossification of the Skull in Cleidocranial Dysostosis. H. Schäfer. Fortschr. a. d. Geb. d. Röntgenstrahlen **85:** 309–316, September 1956. (In German) (Heubnerweg 6, Berlin-Charlottenburg 5, Germany)

Cleidocranial dysostosis is the result of a disturbance of the ossification of the membrane bones, *i.e.*, the bones that develop directly out of fibrous tissue without the mediation of cartilage. The disease is thought to be hereditary and it is assumed that there is a disturbance of an unknown center governing the ossification of the membranous bones.

The disease is often not quite typical. Many cases are reported in which there are also growth disturbances of cartilage bones, particularly of the pelvis, the hips, the spine, etc. On the other hand, the process may be limited to certain of the membrane bones, while others are not affected. The cranial bones, for instance, may be involved but not the clavicles. The author agrees with Rhinehart, who suggests that such cases be called an abortive form of a mutational dysostosis (see Radiology **26:** 741, 1936).

The author reports a case which is interesting because of the unusually severe degree of involvement and because of its early recognition. The patient was first observed at the age of two days. At that time the head gave to the palpating hand the impression of a soft balloon. Only two small shells of thin bone, each the size of a cherry, could be felt in the frontal and parietal regions. The ossification was about that of a fetus in the third month of gestation. The clavicles showed large defects. The author observed this case up to the age of sixteen months. He describes the gradual progress of ossification and illustrates this with serial x-ray films and a sketch.

Six roentgenograms; 1 line drawing.

WILLIAM A. MARSHALL, M.D.
Chicago, Illinois

The Skull in Childhood Myxedema: Its Roentgen Appearance. M. Arias Bellini and Ilda Neves. Am. J. Roentgenol. **76:** 495–498, September 1956. (M. A. B., Canelones 1363, Montevideo, Uruguay)

On the basis of a study of 83 patients, the authors

review the roentgen appearance of the skull in childhood myxedema. Closure of the cranial sutures is delayed. Digital impressions are pronounced. The sella turcica is enlarged in most cases, with a characteristic quarter-circle rounded appearance. The clivus is thickened and vertical and the posterior clinoids are not visualized. The cranial sinuses are delayed in appearance or may be absent. Pneumatization of the mastoids is minimal. Dentition is delayed. The facies is characteristic, with a significant increase of the transverse diameters, a flat forehead, and a short nose with sunken bridge. The angle formed by the nasal bones with the frontal bone is reduced. There is marked prognathism.

One drawing. RICHARD G. LESTER, M.D.
University of Minnesota

Angioma of the Skull. G. Lombardi and G. P. Larini. J. Canad. A. Radiologists **7:** 33–35, September 1956. (G. L., Milan, Italy)

Angiomas of the skull are relatively unusual neoplasms. They are found more commonly in women than in men, in the ratio of three to one, and are seen most frequently in middle age. Any portion of the skull may be involved, but the usual location is in the frontal or parietal region, with predominant involvement of the outer table.

Plain skull films are usually characteristic, showing a fine or coarse honeycomb appearance, with a sharply defined halo of decalcified bone. Angiographic studies afford excellent definition of the vascularization of the neoplasm.

Two cases are reported. The first patient was a 45-year-old woman with a six-year history of a gradually enlarging swelling in the right parietal region originally noticed following minor cranial trauma. Angiograms showed a large "feeder" branch from the middle meningeal artery. The neoplasm was resected without incident. In the second patient, a 52-year-old female, angiographic and encephalographic studies demonstrated a much enlarged middle meningeal artery and the usual extensive vascularization of the tumor. In this case, also, the tumor was excised without difficulty.

Eight roentgenograms; 3 photographs.
JAMES W. BARBER, M.D.
Cheyenne, Wyo.

A Simple Cassette Changer for Cerebral Angiography. Nicholas Wetzel and Robert Masler. J. Neurosurg. **13:** 523–524, September 1956. (Northwestern University Medical School, Chicago, Ill.)

An apparatus which has been used in the Department of Radiology at Passavant Memorial Hospital (Chicago) for several hundred cerebral arteriograms is described. This was constructed in a home workshop from materials costing less than ten dollars.

The apparatus consists of an L-shaped frame with films for the lateral view being held in the vertical position. Lead-backed cassettes are used and are pushed against a Lysholm grid by a spring-backed pressure plate. A series of three 10 × 12-inch lateral films can be obtained as rapidly as the cassettes can be removed, possibly in as little as five seconds. With slight alterations an equal number of anteroposterior films could be taken. After each of the first two exposures, the exposed film is pulled from the apparatus by a tab of adhesive tape previously fixed to the cassette.

While it may be that a greater number of films taken in shorter periods of time is desirable, the authors believe that this apparatus functions better than do many devices costing much more.

One photograph. RICHARD A. ELMER, M.D.
Atlanta, Ga.

Prenatal Obstruction of the Fourth Ventricle. Donald D. Matson. Am. J. Roentgenol. **76:** 499–506, September 1956. (Harvard Medical School, Boston, Mass.)

Accurate diagnosis of prenatal obstruction of the foramina of Magendie and Luschka in the first few months of life is of great importance, since successful surgical treatment is feasible. The diagnosis can be suspected from clinical examination and can be confirmed roentgenologically.

As a result of obstruction of these foramina, the fourth ventricle dilates, especially posteriorly, laterally, and downward into the cervical spinal canal, becoming a huge cyst-like structure. The cisterna magna is obliterated or undeveloped. The mid-line structures of the cerebellum are deficient or entirely absent, while the cerebellar hemispheres are displaced laterally and the flocculus appears rudimentary. The pons, medulla, and upper cervical cord are flat, broad, and displaced anteriorly. The entire tentorium, including its site of attachment to the cranial vault, is displaced upward, carrying with it the transverse sinuses and the torcula. There is symmetrical internal hydrocephalus involving the aqueduct of Sylvius and the third and lateral ventricles.

The clinical appearance is characteristic. In addition to the general signs of hydrocephalus, there is marked prominence of the occipital region. This is in contrast to the appearance in congenital stenosis of the aqueduct of Sylvius, the more common cause of internal hydrocephalus, in which the head is enlarged primarily in the parietal and frontal regions.

Plain skull roentgenograms show an enlarged cranial vault. In addition, there are striking bulging and thinning in the occipital region. The torcula is placed high, and the transverse sinuses are seen to be high and slanting. The lambdoid suture is usually strikingly widened. Ventriculograms show a pathognomonic appearance. All the ventricles are dilated, including the fourth. With the patient in the inverted position, a huge posterior fossa cyst, projecting into the cervical spinal canal to the level of the second cervical vertebra is seen. Dural sinograms, obtained following injection of 3 to 5 c.c. of Diodrast into the longitudinal sinus through the anterior fontanel, confirm the diagnosis, showing the location of the torcula and transverse sinuses.

Surgical treatment consists of suboccipital craniectomy and excision of the cyst wall. The arch of the atlas is removed in order to permit removal of that portion of the cyst projecting into the cervical canal. Early recognition and treatment of this lesion are necessary for satisfactory recovery.

Eight roentgenograms; 5 photographs; 3 drawings.
RICHARD G. LESTER, M.D.
University of Minnesota

Cytomegalic Inclusion Disease with Calcification Outlining the Cerebral Ventricles. George L. Sackett, Jr., and Mary M. Ford. Am. J. Roentgenol. **76:** 512–515, September 1956. (St. Luke's Hospital, Cleveland, Ohio)

Cytomegalic inclusion disease is a rare entity, thought

to be viral in origin and to be transmitted *in utero*. It is characterized by the presence of giant polymorphic cells containing nuclear and cytoplasmic inclusions. These cells are found especially in epithelial structures, such as bronchial epithelium, liver and bile duct cells, and renal tubular epithelium, and less often in the adrenals, pancreas, and thyroid gland, and in the gastrointestinal tract.

Roentgenograms of the skull in the case reported in this paper were noteworthy. In addition to microcephaly and slight overriding of the bones of the calvarium, a distinct layer of calcification following the outline of dilated lateral ventricles could be seen. A film of the abdomen showed hepatomegaly and slight splenomegaly.

Postmortem the brain weighed only 50 gm. after fixation. The cortex was thin and the lateral ventricles dilated. A layer of calcification was found in the brain substance around the ventricles. There was also a small amount of calcium in the basal ganglia.

Only one case of cytomegalic inclusion disease with calcification outlining the lateral ventricles has been previously reported (Mercer, Luse, and Guyton: Pediatrics **11**: 502, 1953. Abst. in Radiology **62**: 466, 1954).

Two roentgenograms; 2 photomicrographs; 2 photographs. RICHARD G. LESTER, M.D.
University of Minnesota

Paratrigeminal Epidermoid Tumors. Carl H. H. Baumann and Paul C. Bucy. J. Neurosurg **13**: 455–468, September 1956. (C. H. H. B., 404 Medical Arts Bldg., Billings, Mont.)

Three cases of paratrigeminal epidermoid tumor are reported, 1 of which was correctly diagnosed preoperatively by a combination of the clinical signs and symptoms and the radiographic changes. The authors believe that these changes collectively give considerable help in differentiation from other lesions in this region, particularly meningiomas and neuromas.

The radiographic features are erosion and destruction of the extreme apex of the petrous pyramid and erosion of the floor of the temporal fossa, with complete destruction in the region of the foramina spinosum and ovale. There is no sclerosis, and the area of destruction is sharply circumscribed.

Clinically the condition was characterized by an insidious onset and complete anesthesia as a result of compression of the trigeminal nerve and gasserian ganglion, without significant pain. Involvement of the motor division of the trigeminal nerve resulted in these patients in paralysis and atrophy of the muscles of mastication, with deviation of the jaw toward the involved side with the mouth open. In addition to the trigeminal nerve, tumors in this location may involve many other structures.

Surgical removal of the epidermoids in the cases reported led to excellent results.

Four roentgenograms; 2 photomicrographs.
RICHARD A. ELMER, M.D.
Atlanta, Ga.

Granular Cell Tumor of the Neurohypophysis. Norman Glazer, Harry Hauser, and Harry Slade. Am. J. Roentgenol. **76**: 324–326, August 1956. (N. G., Children's Hospital, Akron, Ohio)

The authors report the second case of an entity known as granular-cell tumor of the neurohypophysis. Their case duplicated in nearly every aspect the details of the first case reported by Harland (Cancer **6**: 1134, 1953).

The prevailing opinion is that the tumor most likely arises from neural, perineural, or endoneural cells. It expands slowly and attains a considerable size. The optic nerves are compressed, and visual impairment is a predominant symptom. Increased intracranial pressure soon appears, and death follows unless a decompressing procedure is carried out.

Skull roentgenograms demonstrated erosion of the dorsum sellae and posterior clinoids. Pneumoencephalography revealed an elevation of the floors of the right lateral ventricle and the third ventricle. There was no calcification. The tumor in this instance was partially excised. High-voltage roentgen therapy was given postoperatively, but at autopsy there was no evidence of radiation effect.

Three roentgenograms.

ROBERT H. LEAMING, M.D.
Memorial Center, New York

Secretory Sialography in Diseases of the Major Salivary Glands. Irving M. Blatt, Philip Rubin, A. James French, James H. Maxwell, and John F. Holt. Ann. Otol., Rhin. & Laryng. **65**: 295–317, June 1956. (University of Michigan Medical School, Ann Arbor, Mich.)

This report is based on an analysis of 135 cases involving more than 200 sialograms obtained by secretory sialography, the technic of which has been previously described (Ann. Otol., Rhin. & Laryng. **64**: 667, 1955. Abst. in Radiology **67**: 126, 1956).

Inflammatory diseases of the salivary glands are divided into two groups: (1) chronic obstructive sialodochiectasis, in which the abnormalities are primarily in the main duct, and (2) chronic non-obstructive sialodochiectasis, involving chiefly the peripheral ducts.

Calculi and strictures are the principal causes in the obstructive group. The most important sialographic finding is dilatation of the main duct (Wharton's or Stensen's duct) and its primary and secondary ramifications. In cases of long standing there may be a loss of the arboreal pattern of the gland.

Chronic non-obstructive sialodochiectasis or sialectasis includes Mikulicz's disease, Sjögren's syndrome, and recurrent pyogenic parotitis in adults and children. The sialographic pattern shows four stages: punctate, globular, cavitary, and destructive.

Neoplasms of the salivary glands can be identified by sialographic technic as invasive or encapsulated. Infiltrative neoplasms characteristically show disruption of the gland architecture in the filling phase. Secretory function is lost as a result of destruction or replacement by the invasive growth.

The authors conclude that the secretory technic has enhanced the value of sialography as a diagnostic and interpretive aid.

Fifteen roentgenograms; 1 photomicrograph; 2 photographs; 1 table. CADMAN CHAFFIN, M.D.
Mercy Hospital, Pittsburgh, Penna.

Contact Radiography of the Jaw Area. Genyo Yamaguchi and Sadako Yagi. J. Radiol. & Phys. Therapy, Univ. Kanazawa **36**: 220–233, September 1956. (In Japanese, with English abstract) (Department of Radiology, Faculty of Medicine, Kanazawa University, Kanazawa, Japan)

Contact roentgenography is based on the fact that

objects close to the cassette are more sharply defined. In 1936, Miyaji produced contact roentgenograms of the sternum with the use of a Chaoul contact roentgen therapy installation. The innovation was ingenious from a strictly physical point of view, but in 1954 Ott showed that with Chaoul's tube in 1.5 sec., at 44 kv and 2 ma, the skin receives 220 r, while the same roentgenographic result could be obtained with a dental unit in 0.5 sec., at 60 kv and 10 ma, which raised the surface dose to only 6 r.

This paper reports a comparative study of contact roentgenography of the mandible, with particular attention to the temporomandibular joint, first with a phantom, then on patients. With the Chaoul tube (which has a relatively large ring-shaped focus), for a sharply defined picture the object-film distance must be shorter than one-eleventh of the target-film distance, but for adequate blurring of intervening structures the object-film distance must be longer than three-elevenths of the target-film distance. With the dental unit (which has a fine, 1.0-mm. focal spot), the object-film distance must be only shorter than one-fourth of the target-film distance to achieve similar sharpness, while proper blurring is obtained when this object-film distance is longer than two-thirds of the target-film distance. When the dental unit is used, this implies removing the cone.

For contact roentgenography of the mandible, the most favorable target-film distance for frontal projection (central beam directed over the interspace between C-1 and C-2) was found to be 22 cm. with the Chaoul, and 15 cm. with the dental tube, while for the lateral projection (central beam perpendicular to the temporomandibular joint) the best target-film distance appeared to be 36 or 37 cm. with the Chaoul and 19 cm. with the dental tube. The exposure data were: for the Chaoul tube, 80 kv, 4 ma, 4–5 sec. for the lateral and 8–10 sec. for the frontal projection; for the dental tube, 60 kv, 10 ma, 1/10 sec. for the lateral and 1/5 sec. for the frontal projection. Flexible cassettes are highly recommended, with medium-speed screens to preserve detail.

When adequate experience has been accumulated, the use of contact roentgenography (especially with the dental unit) can provide otherwise unobtainable studies of the temporomandibular joint, ramus, maxillary sinuses, orbit, and nasal cavity, without the disturbing shadows of superimposed adjacent structures.

Nine roentgenograms; 12 drawings, two tables.

HYO HYUN BYUN, M.D.
Cook County Hospital, Chicago

Operative Parathyroid Arteriography for Location of Parathyroid Tumour. R. E. Steiner, Russell Fraser, and Ian Aird. Brit. M. J. **2:** 400–401, Aug. 18, 1956. (Postgraduate Medical School of London, London, England)

It is pointed out that the operative discovery of a parathyroid tumor which lies at a distance from the thyroid gland, and especially of a parathyroid tumor in the superior mediastinum, can be a difficult surgical exercise. Any procedure which locates such a tumor offers a real advantage. It has been found possible to outline a parathyroid tumor by injection of contrast substance through a catheter in the aorta, but with this technic some cerebral complications have occurred.

The authors present a case in which visualization of the parathyroid gland was obtained by injection of contrast substance into one of the inferior thyroid arteries. The larger of the two arteries was chosen for injection. During the earlier phase of the operative procedure, the superior thyroid vessels had been visualized and were found to be symmetrical on the two sides.

The injection was performed by means of a "polythene" tube with a hypodermic needle. The artery to be injected was elevated on a thread support at the point of its emergence from under cover of the carotid sheath, with just enough tension to stop the flow in it. The needle was then inserted into the lumen of the artery just distal to the point of its occlusion, and the radiopaque material was rapidly introduced. Three films were obtained. The first, at the end of the injection, showed an opacified area below and somewhat lateral to the inferior pole of the thyroid gland. This proved to be the site of the parathyroid tumor, which was successfully removed.

It is pointed out that the disparity in size between the paired thyroid arteries will determine the proper site of injection. The character of the shadows suggests that if the parathyroid tumor had lain on the side of the trachea, or in the groove between the trachea and esophagus, the shadow of the thyroid gland and parathyroid tumor would not have been separately distinguishable. However, in this situation, there should be no difficulty in operative dissection without the aid of x-ray visualization.

One roentgenogram.

RICHARD A. ELMER, M.D.
Atlanta, Ga.

Three Unusual Cases of Hyperparathyroidism. D. J. M. Crawford, J. Stefanelli, and A. F. Alvarez. Brit. J. Surg. **44:** 193–202, September 1956. (C. S. Williams Clinic, Trail, British Columbia)

Three cases of hyperparathyroidism are reported, each of which displayed some feature out of the ordinary. In the first case the unusual feature was the youth of the patient, who was seen at the age of three years, with a limp which had its onset a year earlier. A roentgenogram showed a well defined circular area of rarefaction in the trochanteric region of the left femur, which was interpreted as a simple bone cyst and diagnosed on operation as osteitis fibrosa cystica. The child did well for five months and then began to limp anew. Roentgenograms of the long bones showed no further cysts but an abnormal degree of rarefaction appeared to be present. In view of this observation and repeated laboratory studies a diagnosis of hyperparathyroidism was considered likely and exploration was undertaken. The only evidence of parathyroid tissue found was a small pinkish structure, measuring 0.5 × 0.3 cm., which was removed. The histologic diagnosis was parathyroid adenoma.

In the second case recurrent hyperparathyroidism and recurrent peptic ulceration were closely associated.

The third patient was originally operated upon for a swelling in the premaxillary area, which was considered to be an osteoclastoma. This diagnosis suggested the possibility of hyperparathyroidism. Roentgenograms showed marked osteoporosis with scattered small cysts, the lamina dura of the teeth was absent, and there was calcification of the blood vessels and some of the soft tissues in the limbs. The blood chemistry further supported a diagnosis of hyperparathyroidism, and exploration of the neck revealed a small pale structure about 1.5 cm. in diameter below the thyroid. The histological

diagnosis was malignant adenoma of the parathyroid gland. Postoperatively the patient was treated with deep x-ray therapy (3,500 r). Six months later she was re-admitted with severe anemia and in spite of vigorous therapy expired.

Carcinoma of the parathyroids is extremely uncommon. In this last case the histologic findings were in accord with such a diagnosis, and the postmortem study suggested the possibility of a local recurrence, but in the absence of metastases the diagnosis cannot be said to be proved.

The authors follow their case presentations with a consideration of some of the clinical findings, especially in early stages of the disease.

Early diagnosis of hyperparathyroidism is important for the prognosis. Death from renal failure is common. The importance of recognizing the co-existence of peptic ulceration in cases of hyperparathyroidism is emphasized anew.

Six roentgenograms; 11 photomicrographs.

JOACHIM GROELLER, M.D.
Cleveland City Hospital

Obstructed Endotracheal Tube Demonstrated by Roentgenogram. Richard C. Thompson. J.A.M.A. **162:** 194–196, Sept. 15, 1956. (545 Fairfax Ave., San Mateo, Calif.)

The author has encountered 2 cases of partial obstruction of endotracheal anesthesia tubes, in which roentgenograms were helpful in demonstrating the cause of the difficulty. In each case the bevel of the tube was found to be resting against the anterior wall of the trachea. In either the dorsal or the prone position, when the head is turned sharply to the side, as in certain orthopedic, intracranial, or otological operations, the hazard of obstruction due to this cause may occur. If the neck is markedly extended, the trachea will curve posteriorly and a slight rotation of the tube may result in obstruction at the bevel. Use of a fenestrated tube is suggested to make endotracheal anesthesia a safer procedure in this respect.

Four roentgenograms; 9 diagrams.

JOHN P. FOTOPOULOS, M.D.
Hartford, Conn.

THE CHEST

Ventilatory and Lung Volume Determinations in Patients with Chest Deformities. Harry N. Iticovici and Harold A. Lyons. Am. J. M. Sc. **232:** 265–275, September 1956. (Cardiopulmonary Laboratory, State University of New York College of Medicine at New York, N. Y.)

The authors present the results of a study of the maximum breathing capacity and vital capacity of 7 patients with thoracic deformities. The results indicate a reduction of one or both of these values in all cases. Patients with kyphoscoliosis and Marie-Strümpell disease showed a lowering of both values, with an increase in the ratio of residual volume to total lung capacity. In pes excavatum vital capacity was normal.

The abnormalities found are characterized by a decreased total lung volume due to decreased expansibility of the chest wall with an accompanying restrictive breathing pattern.

Eight roentgenograms; 2 graphs; 1 table.

THEODORE E. KEATS, M.D.
University of Missouri

Congenital Cystic Adenomatoid Malformation of the Lung in Infants. John M. Craig, John Kirkpatrick, and Edward B. D. Neuhauser. Am. J. Roentgenol. **76:** 516–526, September 1956. (Harvard Medical School, Boston, Mass.)

Four proved cases of congenital cystic adenomatoid disease of the lung in infants are described in detail. Clinically these patients were cyanotic, with rapid respirations, and absence of respiratory distress and fever. The physical signs were those of mediastinal displacement to the side opposite that of involvement, dullness to percussion over the affected lung, and diminished breath sounds over this area.

Roentgenologically all 4 cases showed an intrapulmonary mass of soft-tissue density, containing scattered radiolucent areas irregular in size and shape. The mass was sharply circumscribed and was limited to one lobe. The heart and mediastinum were displaced to the opposite side.

Anatomical specimens showed a lack of the usual septation of the affected areas into discrete lobules and absence of a well defined intrapulmonary bronchial system. The failure of cartilage formation in these large collapsible structures may play a role in their great size and their apparent distention. Absence of the usual lobular septation allows diffusion of air into all parts of the anomalous portions of the lung.

Since in 2 of the cases the involved lung rapidly increased in size, with a concomitant increase in the air-containing cystic structures with further displacement of the mediastinum and compromise of respiratory function, it appears that, once a definite diagnosis of such adenomatoid cystic malformations is made, surgery is mandatory.

In true congenital cystic disease of the lung the large cysts are lined by cartilage-containing walls and there is usually no roentgenologically demonstrable mass. Congenital cystic adenomatoid malformation should not be confused with an accessory lung, which has no connection with the bronchial system or pulmonary blood supply, or the sequestrated lobe connected usually to the bronchial system but deriving its blood supply from a branch of the aorta. The differential diagnosis includes also the chondromatous hamartomas and angiomatous malformations. Hamartomas are more frequent in adults but they have been described in the newborn.

The authors conclude that adenomatoid malformation of the lung in early infancy is a form of true congenital cystic disease of the lung and early extirpation of the lesion is the proper treatment.

Eight roentgenograms; 5 photographs; 2 photomicrographs; 1 table.

JOHN P. FOTOPOULOS, M.D.
Hartford, Conn.

A Clinical Review of Pulmonary Microlithiasis. Heinz H. Meyer, Earl S. Gilbert, and Geoffrey Kent. J.A.M.A. **161:** 1153–1157, July 21, 1956. (H. H. M., 7501 W. Cermak Road, Berwyn, Ill.)

A form of diffuse pulmonary calcification called microlithiasis alveolaris pulmonum has been reported sporadically in Europe and more recently in the United States. The authors present the clinical findings in a case of their own and review the 16 cases appearing in the literature.

The disease is characterized by a latent asymptomatic period which has been known to last as long as twenty-five years. During this time the most impres-

sive clinical feature is the roentgen appearance of minute nodules disseminated throughout all portions of the lung fields, with greater density toward the bases, where eventually cardiac and diaphragmatic outlines are obscured. These findings represent innumerable intra-alveolar calcospherites. In later stages, pulmonary interstitial fibrosis appears, with gradual decrease in vital capacity. Eventually, cor pulmonale and cardiac decompensation supervene.

All diseases showing disseminated pulmonary nodulation must be considered in the differential diagnosis. Close correlation with the clinical findings helps rule out many of these conditions.

There are not as yet sufficient observations to warrant definite conclusions as to etiology of pulmonary microlithiasis but the authors feel that it may be the result of a hyperimmune inflammatory response to one or, more likely, a variety of irritants. The resulting peculiar alveolar exudate is not easily absorbed and may undergo organization and calcification. Once present, the disease gives no evidence of regression.

Two roentgenograms; 2 photomicrographs.

ARTHUR SHUFRO, M.D.
University of Michigan

Varicella Pneumonia: Case Report. Ray G. Cowley, William E. Crew, and Gerard R. Hassett. Ann. Int. Med. **45:** 518–525, September 1956. (R. G. C., P. O. Box 115, William Beaumont Army Hospital, El Paso, Texas)

A case of primary varicella pneumonia in a 33-year-old Hawaiian male is reported. There was a history of exposure to varicella and a typical eruption was present. The clinical and laboratory features were consistent with those described in previously recorded cases. Initially mild respiratory symptoms became severe on the sixth day. A roentgenogram of the chest revealed an extensive bilateral, nodular, and interstitial-like infiltration. Because of severe respiratory embarrassment, the patient was kept in an oxygen tent for forty-eight hours. At the end of that time respiratory function was rapidly improving, and the varicella skin lesions began to heal. Serial roentgenograms showed gradual clearing, with complete resolution of the diffuse pulmonary infiltration by the twenty-fifth day.

There was no evidence that Aureomycin was effective in altering the course of the disease process in this case. It is probable that the only value of antibiotics in varicella infections is prophylaxis against secondary bacterial invaders. Because of the serious nature and the severity of this complication, such prophylactic use of antibiotics is not without merit.

Three roentgenograms.

Solitary Circumscribed Lesion of the Lung Due to Cryptococcus Neoformans. Charles T. Pinney. Am. Rev. Tuberc. **74:** 441–444, September 1956. (201 S. Union St., Olean, N. Y.)

A case of torulosis with involvement limited to the lung is reported. The lesion, presenting as a solitary nodule in the anterior segment of the right upper lobe, was found on a routine chest roentgenogram taken on discharge from military service. The patient had had an episode of respiratory infection two years earlier, at which time a lesion of similar appearance was demonstrated in the same location. This nodule was believed to have increased slightly in the two-year interval. An anterior segmental resection was carried out, revealing a well encapsulated, caseous granuloma with daughter foci. Culture showed the typical organisms of Cryptococcus neoformans.

From this observation it appears that cryptococcosis may take the form of asymptomatic nodular lesions similar to those noted in other chronic granulomata, though it usually occurs in association with central nervous system involvement. Roentgenographic findings are not characteristic, and single or multiple cavitary lesions, pleural effusion, pneumonic infiltration, empyema, and dense consolidations have all been described. There seems to be a predilection for the lower lobes. Diagnosis depends upon bacteriological study, since radiographic findings are similar to those in other chronic granulomatous diseases.

Four roentgenograms.

J. H. JUHL, M.D.
University of Wisconsin

Pulmonary Congestion and Edema in Uremia. Skottowe W. DePass, Joseph Stein, Maxwell H. Poppel, and Harold G. Jacobson. J.A.M.A. **162:** 5–9, Sept. 1, 1956. (J. S., 130 W. Kingsbridge Rd., Bronx 6, N. Y.)

The pulmonary roentgenographic picture observed in patients with uremia has been described in the past as "pulmonary azotemia," "uremic lung," or "uremic pneumonia." It is characterized by symmetrical central infiltrations in each lung with a comparatively clear peripheral zone in both pulmonary fields. The purpose of the present study is to deny the existence of such a picture as a roentgenographic entity limited to cases of uremia. Actually it represents pulmonary congestion and edema following fluid retention either on a cardiac or a renal basis.

Six cases are presented, in each of which the typical roentgen appearance described above was found. Only 3 of the patients had uremia, but all had symptoms consistent with congestive heart failure. Fluid retention, varying degrees of cardiac failure, superimposed pulmonary infection, as well as an increased respiratory rate, all enter into the formation of the picture.

The authors believe that the primary cause of the changes seen on chest roentgenograms of the patients with renal disease presented here was water and salt retention as the result of the disturbance of renal function. The same roentgenographic changes can be produced by primary cardiac decompensation without renal disease.

Six roentgenograms; 1 table.

JOHN F. RIESSER, M.D.
Springfield, Ohio

Correlation of Tomographic and Bronchographic Findings in Apical Bronchiectasis. J. H. Juhl, W. J. Alt, and R. H. Wasserburger. Am. Rev. Tuberc. **74:** 388–399, September 1956. (Department of Radiology, University Hospitals, Madison 6, Wisc.)

In an attempt to evaluate the usefulness of tomograms in determining the presence or absence of apical bronchiectasis in patients with pulmonary tuberculosis, the authors studied the films of 100 patients in whom tomography was followed by bronchography. Only patients in whom bronchographic mapping was complete were included in the series. The tomograms were read without reference to previous films, to bronchograms, or to clinical history.

In 18 patients with minimal tuberculosis, 5 errors were made on the basis of the tomograms. Fifty-three

of the patients had moderately advanced tuberculosis and comparable degrees of bronchiectasis were found tomographically and bronchographically in 25. In 23 patients the tomographic interpretation was in error. In 5, neither examination showed bronchiectasis. The correlation was much better in far advanced tuberculosis, comparable degrees of bronchiectasis being found in 20 of 22 instances.

There was a tendency to over-read the tomograms in minimal disease and to under-read them in moderately advanced cases. Bronchiectasis could be readily recognized in far advanced contracted and fibrotic areas because the thickness of the bronchial walls plus peribronchial infiltrate resulted in visualization of bronchi well out in the parenchyma and made their identification relatively easy. Bronchiectasis was missed in the moderately advanced disease where there was not enough thickening of the bronchial walls to permit visualization and identification of bronchi far enough toward the periphery to evaluate their caliber. In other instances, parallel strands of fibrosis or vascular markings simulating bronchial walls were misinterpreted as dilated bronchi to produce over-reading.

The study indicates that, while tomography is useful in the diagnosis of bronchiectasis associated with tuberculosis, it is not accurate enough to furnish reliable information regarding the condition of bronchi in the presurgical evaluation of tuberculous patients. When bronchography is contraindicated, however, tomograms can be a useful substitute. They are superior to bronchograms in outlining the thickness of bronchial walls.

Nineteen roentgenograms; 1 table.

J. H. JUHL, M.D.
University of Wisconsin

"Silo-Filler's Disease"—a Syndrome Caused by Nitrogen Dioxide. Thomas Lowry and Leonard M. Schuman. J.A.M.A. **162**: 153–160, Sept. 15, 1956. (T. L., 629 Medical Arts Bldg., Minneapolis 2, Minn.)

The term "silo-filler's disease" has been used to designate any bronchial or pulmonary condition produced by the inhalation of oxides of nitrogen derived from fresh silage. The authors report in detail 4 cases of one form of the disease, typical bronchiolitis fibrosa obliterans, occurring in individuals exposed to silage fumes under identical circumstances. This entity produces a striking clinical syndrome, but the roentgenographic findings may be indistinguishable from those of other well established pulmonary diseases. A history of recent exposure to irritating silage fumes is the essential evidence that identifies the condition.

The following cardinal points noted in all 4 of the authors' patients are considered characteristic of the clinical picture of silo-filler's disease with bronchiolitis obliterans. (1) There is a history of inhalation of irritating gas in or about a silo within a short time (from a few hours to three or four days) after filling is commenced. (2) Cough and dyspnea with a sensation of choking and severe weakness are noted immediately upon exposure and do not relent completely during the illness. (3) There is a two- to three-week period after exposure characterized by relative remission of symptoms, although some cough, malaise, and dyspnea remain and weakness progresses. (4) Onset of a second phase of illness occurs about three weeks after initial exposure, often accompanied by chills. There is fever, with progressively more severe dyspnea, cyanosis, and cough which do not respond to administration of antibiotics, oxygen, or bronchodilators and lead to death (or else to the beginning of recovery) three and one-half to six weeks after exposure. (5) Numerous fine and medium moist inspiratory râles are heard over the entire extent of the lungs together with a few moist expiratory râles and prominence of sibilant asthmatic type râles during expiration (signifying obstruction of small bronchi and bronchioles). (6) Roentgen examination of the chest reveals uniform infiltration of all lung tissue with innumerable discrete nodular densities. The roentgenogram cannot be distinguished from that of acute miliary tuberculosis or any one of the other conditions producing diffuse bilateral nodulation in lungs. Confluence of the lesions may be observed in severe and advanced cases. (7) Laboratory studies show moderate to marked neutrophilic leukocytosis. In the late stages of the disease, progressive increase of blood carbon dioxide levels, indicating retention of this gas in spite of extreme respiratory effort, is a poor prognostic sign. Results of other laboratory tests are normal; cultures of blood, sputum, and bone marrow for bacteria and fungi and virus studies have all been negative.

Two of the authors' cases were fatal, and autopsy showed typical lesions of bronchiolitis fibrosa obliterans. It was established that sufficient amounts of nitrogen dioxide are produced in freshly filled silos to account for the bronchial disease encountered.

Six roentgenograms; 4 photomicrographs.

JOHN P. FOTOPOULOS, M.D.
Hartford, Conn.

Roentgen Observations after Perforation of Lymph Nodes into Bronchi in the Course of the Primary Infection. G. Voluter. J. de radiol. et d'électrol. **37**: 738–749, September-October 1956. (In French) (Université de Genève, Geneva, Switzerland)

Caseating lymph nodes may perforate into a bronchus during the course of the primary infection or in later stages of tuberculosis in which other factors are present leading to decreased resistance to the disease. The pressure of enlarged nodes may act directly to produce atelectasis or, after perforation, the caseous masses may lead to atelectasis or a localized tuberculous pneumonia or may disseminate to form numerous small tuberculous foci. In some patients the enlarged nodes are radiologically evident and suggest directly the course of events; in others the actual region of perforation may be obscure, the signs pointing indirectly to the pathogenesis.

Sixteen roentgenograms; 1 photograph.

CHARLES M. NICE, JR., M.D., PH.D.
University of Minnesota

THE HEART AND BLOOD VESSELS

High-Speed Cineradiography and Electrocardiography; a New Method for the Study of Heart Disease in Humans. Myron Prinzmetal, Rexford Kennamer, Stanley M. Weiner, and John R. Bishop. J.A.M.A. **161**: 1229–1231, July 28, 1956. (M. P., 4751 Fountain Ave., Los Angeles 29, Calif.)

The authors describe their method of simultaneously recording high-speed cineradiograms and electrocardiograms. The electrocardiogram is obtained by tapping the EKG apparatus and leading it to an oscilloscope, the output of which is led to a long-persistence cathode-

ray tube. By a system of lenses the electrocardiographic and intensified fluoroscopic images are superimposed on the camera film. With the use of slow motion for projection, detailed analysis of the motion of the cardiac borders can be made, with the establishment of the exact point in the cardiac cycle.

Modifications of the apparatus would allow simultaneous recording of cardiac catheterization pressures, angiocardiograms, ballistocardiograms, or phonocardiograms instead of electrocardiograms.

One roentgenogram; 2 drawings.

ARTHUR S. SHUFRO, M.D.
University of Michigan

Annular Sclerosis of the Atrioventricular Septum and Calcification of the Heart Valves; a Contribution to the Differential Diagnosis of Intracardiac Calcifications. W. Milatz. Fortschr. a. d. Geb. d. Röntgenstrahlen **85:** 282-292, September 1956. (In German) (Institut für Röntgenologie und Radiologie der Charité Berlin, Berlin, Germany)

The atrioventricular septum of the heart is the plane of connective tissue structures separating the musculature of the ventricles from that of the atria. It contains the valves of the heart. If an x-ray beam is directed through the chest in a sagittal direction, this plane will be projected into the left paravertebral area at the level of the third intercostal space and will be visualized, providing there is enough calcium content in the valves and that the rays are hard enough (preferably 120 kv). Calcification in the valves is quite common (5.4 per cent of autopsies) but is often overlooked because it can be demonstrated only if very hard rays are used.

The calcification may occur as a circular ring due to calcium deposits in the annulus fibrosus, or may involve the valves themselves. During fluoroscopic examination (with hard rays and narrow diaphragm) calcific deposits may be identified by their characteristic "dancing" movements. It has been demonstrated by kymographic examinations that in systole the contraction of the ventricular muscle alone is not sufficient to eject the usual volume of 100 to 120 c.c. of blood; the efficiency of the muscle action of the ventricle is greatly augmented by a pump-and-piston mechanism of the atrioventricular septum, whereby this plane moves toward the apex of the heart, accounting for the major part of the volume of blood that is being propelled. In diastole this plane retracts. The amplitude of these movements amounts to 2 or 3 cm. in a healthy individual, as was plainly demonstrated by cinematographic studies.

Most calcifications are found during fluoroscopic examination. The following criteria are postulated as proof that the calcium deposits are in the valves and not elsewhere. First, the calcification must remain within the heart shadow in all planes of projection. Second, it must not change its location relative to the heart during respiration. Third, the pulsating movements of the calcification must be greater than that of the left border of the heart. Fourth, the maximal amplitude of movement must be demonstrable in the first oblique projection.

Most of the calcifications are attributable to either inflammatory or degenerative processes.

Six roentgenograms; 1 photograph; 4 line drawings.

WILLIAM A. MARSHALL, M.D.
Chicago, Ill.

Localized Interlobar Effusion in Heart Failure: Phantom Lung Tumor. Bernard H. Feder and Stefan P. Wilk. Dis. of Chest **30:** 289–297, September 1956. (B. H. F., Veterans Administration Hospital, Long Beach, Calif.)

"Phantom lung tumor" is a localized interlobar transudate frequently encountered in congestive heart failure. On a postero-anterior film, the localized effusion presents a dense, homogeneous appearance; it is sharply demarcated, round, oval, spindle or kidney-shaped, resembling a pulmonary tumor. The lateral projection localizes the density to the fissure. The usual site of the effusion is the lesser fissure on the right side. Multiple localized collections are rare. Associated findings are cardiac enlargement, vascular congestion, and occasionally a general pleural effusion. The characteristic finding is the disappearance of the "tumor" on digitalization and diuresis. The density may reappear in the same location in a subsequent bout of failure.

The pathogenesis is not clear. Among the theories which have been advanced are (1) adhesions in the pleural cavity due to antecedent infection; (2) repeated bouts of heart failure with hydrothorax, leading to a pleural reaction with adhesions, with the interlobar fissures affected the least; (3) the residual of a general pleural effusion, with encapsulation because of the deposition of fibrin.

The authors give 4 short case histories with illustrations showing the interlobar collections of fluid and the disappearance of the phantom tumors.

Fifteen roentgenograms.

HENRY K. TAYLOR, M.D.
New York, N. Y.

The Radiologic Diagnosis of Coronary Insufficiency. Ismet Sayman. J. de radiol. et d'électrol. **37:** 783, September-October 1956. (In French) (Denizcilik bankasi hastanesi, Röntgen mütehassisi, Istanbul-Galata, Turkey)

In this brief paper a relatively simple method is described for the radiological diagnosis of coronary insufficiency. Fluoroscopy and roentgen kymography are performed before and after submitting the patient to effort. During fluoroscopy, and on the ensuing kymogram, special attention is paid to the left ventricle in the postero-anterior and left anterior oblique positions. In the areas involved by coronary insufficiency there is a diminution in the amplitude of pulsations, while in the uninvolved areas pulsation is augmented after effort.

Three drawings.

CHARLES M. NICE, JR., M.D., PH.D.
University of Minnesota

Coarctation of the Aorta. W. P. Cleland, T. B. Counihan, J. F. Goodwin, and R. E. Steiner. Brit. M. J. **2:** 379–390, Aug. 18, 1956. (Postgraduate Medical School of London, London, England)

The authors present an excellent clinical evaluation of 52 patients with coarctation of the aorta. Forty of the patients in this group were operated upon and there were 3 operative deaths.

Most of the clinical aspects of this condition are considered and many interesting features are pointed out. Coarctation is often asymptomatic and 43 of the cases in this series were diagnosed during medical examination carried out for reasons unrelated to the coarctation: heart murmurs in 26, hypertension in 12, pulsating

swelling in the neck in 2, and x-ray observations in 3.

It is pointed out that there is considerable evidence to indicate that most patients with coarctation die before the fifth decade. Causes of death are considered in detail.

In 40 of the patients in this series, radiographs were available for study. Deformity of the aortic arch is considered an important radiological sign. In this series the arch was abnormal in 27 cases—large or aneurysmal in 4, absent in 8, small in 5, and double in 10. Dilatation of the ascending aorta was found in 15 cases and was of aneurysmal proportions in one. The descending aorta displaced the barium-filled esophagus forward in 22 cases. This valuable sign, best seen in the left anterior oblique view, was present in 6 of the 13 cases in which the aortic arch appeared otherwise normal and raised to 33 the number of cases in which roentgen examination without vascular contrast media permitted location of the site of coarctation in the aortic arch. The forward kink of the aorta at the point of coarctation and post-stenotic dilatation caused the displacement of the esophagus. In 2 cases, aneurysm of a post-stenotic intercostal artery produced a shorter indentation of the esophagus.

Notching of the lower rib margins was observed in 31 cases. In 5 instances rib notching was present on one side only. Left ventricular enlargement was demonstrated in 30 cases.

Angiocardiograms were made by the venous route in 14 cases and the site of coarctation was outlined in all of them. The length of the coarctation and the diameter of the distal aorta, however, were often overestimated. As a result, prediction from angiograms that grafts would probably be necessary was open to error. Aortograms made in 2 cases by catheterization of the radial artery gave an excellent outline of the coarctation, surpassing in definition the angiograms obtained by the venous route.

The pros and cons of operation are discussed and summarized and the indications for surgery are evaluated.

Five roentgenograms; 5 other figures; 2 tables.

RICHARD A. ELMER, M.D.
Atlanta, Ga.

Perforation of a Dissecting Aneurysm of the Aorta into the Esophagus. M. Ramseyer. Radiol. clin. **25:** 297–301, September 1956. (In French) (Hôpital de l'Institution des diaconesses de Saint-Loup, La Sarraz, Switzerland)

Only 15 cases of perforation of the esophagus by an aortic aneurysm have been published. The rupture can take two forms, each with its own symptoms: (1) immediate fatal perforation due to simultaneous rupture of the aneurysm and the entire thickness of the esophageal wall; (2) delayed perforation, in which the esophageal wall initially is dissected longitudinally, followed later by mucosal tear and massive hemorrhage into the lumen. The site of involvement of the esophagus is usually in that segment from two or three fingerbreadths below the carina to two or three fingerbreadths above the cardia.

In the delayed variety, the blood dissects the esophageal wall, usually downward, and may even reach the stomach. Dissection upward from the site of perforation is rarer and less extended. During the phase of extension, the patient experiences agonizing retrosternal pain, dysphagia, and cough, but does not vomit blood. Perforation of the esophageal or gastric mucosa occurs some days to weeks after the rupture of the aneurysm.

The author reports a case of an arteriosclerotic dissecting aneurysm of the thoracic aorta with rupture into the esophagus.

Two roentgenograms; 1 drawing.

CHRISTIAN V. CIMMINO, M.D.
Fredericksburg, Va.

Superior Vena Cava Draining into Left Atrium. Another Cause for Left Ventricular Hypertrophy with Cyanotic Congenital Heart Disease. Herman Tuchman, John F. Brown, John H. Huston, Arvin B. Weinstein, George G. Rowe, and Charles W. Crumpton. Am. J. Med. **21:** 481–484, September 1956. (Cardio-Pulmonary Research Laboratory, University of Wisconsin Medical School, Madison, Wisc.)

A case of cyanotic congenital heart disease with left ventricular hypertrophy is described. Cardiac catheterization revealed a superior vena cava which drained into the left atrium. There was no other cardiovascular abnormality. Angiocardiography was not carried out in this case because of fear of presenting a high concentration of contrast material directly to the coronary arteries and systemic circulation and because a satisfactory diagnosis had been made by catheterization. The diagnosis was confirmed by operation and at necropsy.

This case is considered of particular interest because it represents another type of cyanotic cardiovascular anomaly producing left ventricular hypertrophy. The quantity of shunt, as calculated, confirms the existing impression that approximately one-third of the venous return is *via* the superior vena cava and two-thirds *via* the inferior vena cava. The diagnosis should easily be made by means of cardiac catheterization and angiocardiography, and surgical correction of the anomaly is feasible.

Two roentgenograms; 2 photographs; 1 drawing; 1 table.

Coarctation of the Pulmonary Artery. John E. Coles and Weldon J. Walker. Am. Heart J. **52:** 469–473, September 1956. (Brooke Army Hospital, Fort Sam Houston, Texas)

The author gives the history of a twenty-six-month-old white girl in whom a diagnosis of coarctation of the pulmonary artery was made by angiocardiographic and cardiac catheterization studies. Pregnancy and delivery were normal. A cardiac murmur was noted at birth, and the child's color was considered dusky when she cried, but there was no marked cyanosis.

The principal findings were a harsh Grade 4 systolic murmur in the pulmonic area with an associated thrill. Fluoroscopy showed the heart size to be at the upper limits of normal with slight prominence of the main pulmonary artery and normal to decreased vascularity in the peripheral lung fields. An electrocardiogram revealed right ventricular hypertrophy. A diagnosis of valvular pulmonary stenosis with a possible associated interatrial septal defect was made.

Cardiac catheterization with continuous pressure recording revealed a sudden rise in pressure on withdrawal of the catheter as the tip passed from the right pulmonary artery into the main pulmonary artery. Pressure recordings could not be obtained on the left side. An angiocardiographic examination revealed a

normal sequential filling of the cardiac chambers without early opacification of the aorta. The radiologist reported: "At the region of the bifurcation of the pulmonary artery, the diameter of the lumen measures approximately 8 mm. on the right and 10 mm. on the left side. About 1.5 cm. distal to this point the right pulmonary artery measures 14 mm. and the left 12 mm."

The author cites 3 cases reported by Søndergaard (Danish Med. Bull., **1**: 46, 1954) and mentions a fourth case recorded by Shumacker and Lurie (J. Thoracic Surg. **25**: 173, 1953). Dr. F. J. Hodges, at a panel discussion on cardiovascular diseases, at the meeting of the American Medical Association in June 1955, presented a case with similar cardiac catheterization findings. The present case thus appears to be the sixth on record.

Three roentgenograms; pressure curves.

HENRY K. TAYLOR, M.D.
New York, N. Y.

Roentgen-Anatomic Study of a New Shadow Caused by the Pulmonary Veins, and Seen in the Latero-Lateral View of the Chest. B. Passariello and G. Giuliani. Radiol. med. **42**: 841–851, September 1956. (In Italian) (Istituto di Radiologia del Policlinico Umberto I, Ospedali Riuniti di Roma, Rome, Italy)

A round opacity can frequently be noted in the latero-lateral view of a normal chest. It has the appearance of a nodule or of several round, rather confluent shadows located a few centimeters below the hilus. This finding, sometimes interpreted as representing a pathological condition in the hilar region, was re-evaluated by the authors in anatomic-radiologic studies on the cadaver after injection of contrast material into the hilar vessels.

It was found that the pulmonary veins, near their openings into the left auricle, have a definite transverse position, below the main branches of the pulmonary artery. The nodular shadows described represent the projection of the paramedial portion of these veins, especially of the lower branch of the right pulmonary vein. The shadows are thus physiologic and must be considered part of the normal pulmonary hilus.

Twenty-three roentgenograms.

R. G. OLIVETTI, M.D.
Newington, Conn.

Venous Communications Between the Cardiac Veins and the Large Venous Trunks in the Superior Part of the Mediastinum. Carl-Olof Ovenfors. Acta radiol. **46**: 518–522, September 1956. (Karolinska Sjukhuset, Stockholm, Sweden)

In a 10-year-old boy undergoing selective angiocardiography, the tip of the catheter was inadvertently placed in the coronary sinus during injection of the contrast medium. This resulted in filling of the cardiac veins and showed extensive communication with the venous system of the superior anterior portion of the mediastinum.

As such an anastomosis had not been previously described, further investigation was done on autopsy material. In 11 cadavers the coronary sinus was injected following occlusion of the tricuspid orifice and great veins with tampons. Nine such injections were technically successful and demonstrated extensive venous anastomosis similar to that observed in the patient described above. Communication with the left innominate vein *via* the first intercostal vein was demonstrable in some cases, and with the superior vena cava near the opening of the azygos vein in others.

As this anastomosis is not demonstrated on filling of the large veins, but is seen only with forced retrograde injection through the cardiac veins, it is probable that blood flow through this system is unappreciable under normal conditions.

Nine roentgenograms.

J. A. GUNN, M.D.
Grand Rapids, Mich.

Arteriography of the Hand. A. Dimtza. Radiol. clin. **25**: 305–320, September 1956. (In German) (Zurich, Switzerland)

Arteriography of the hand can be of great help in the recognition and handling of diseases of the arteries themselves as well as of post-traumatic conditions. Indefinite clinical states in the hands and fingers, such as thickening, pallor, and coldness, are often explained by the arteriographic findings. The extent of the diseased arterial segment and the measure of the collateral circulation give the points of differential diagnostic importance in chronic edema, necrosis in the fingers, and Raynaud's syndrome. In post-traumatic arterial disturbances, arteriography helps to differentiate spasm from actual obliteration.

Different types of arterial wall injury may arise after trauma, depending upon its localization in the hand and its nature and degree. Especially vulnerable are those arteries that cannot be displaced from their bony base, such as those of the carpus. If permanent arterial spasm follows contusion, the roentgenogram will show a characteristic local tuft formation (small thrombosis and collaterals, small aneurysm ?) at the site of injury, most often near the superficial ulnar arch. Occasionally these changes are either improved or cured through arteriography alone.

The interpretation of arteriograms of the hand and fingers is not always simple. The increased tendency of the entire upper extremity to vascular spasm, especially in the fingers, and the rapid venous filling can lead to false conclusions. Complete agreement of the local clinical findings with the arteriographic observation is essential.

The injection is usually done by open exposure of the brachial artery above its division.

Excellent arteriograms, obtained in an experience of over twenty years, depict the findings in endangiitis obliterans, arteriosclerosis, trophedema, Raynaud's syndrome, and post-traumatic disturbances.

Thirteen roentgenograms; 8 photographs.

CHRISTIAN V. CIMMINO, M.D.
Fredericksburg, Va.

THE BREAST

Transsternal Phlebography of the Internal Mammary Vein. F. Kink. Radiol. clin. **25**: 301–305, September 1956. (In German) (Universitäts-Röntgeninstitut Basel, Basel, Switzerland)

A knowledge of the state of the internal mammary lymph nodes in the clinical evaluation of a patient with carcinoma of the breast is of prime importance. Visualization of the internal mammary veins is of great help toward this end. This can usually be accomplished by the transsternal route. The contrast material is injected into the lower third of the sternum, and the internal mammary veins are rapidly visualized, the whole examination taking only a few seconds.

The abnormalities in the veins may take the form of circumscribed extrinsic defects, displacement and narrowing of a segment with a prominent collateral circulation, or even complete obstruction. The author admits that there are many opportunities for error in diagnosis, such as anomalies, unequal filling, and the presence of a normal venous pattern in spite of metastasis. He believes, however, that the method is of value in the preoperative evaluation of the patient with carcinoma of the breast. His experience includes 31 examinations. The report is to be considered a preliminary one.

Three roentgenograms; 1 photomicrograph.

CHRISTIAN V. CIMMINO, M.D.
Fredericksburg, Va.

HERNIA

Hiatal Hernia Complex. Hiatal Hernia, Peptic Esophagitis, Mallory-Weiss Syndrome, Hemorrhage and Anemia, and Marginal Esophagogastric Ulcer. Felix G. Fleischner. J.A.M.A. **162**: 183–191, Sept. 15, 1956. (330 Brookline Ave., Boston 15, Mass.)

The author states that no intrinsic occlusive mechanism exists at the anatomic cardia of the stomach. Even in cardiospasm or achalasia of the esophagus the obstructing narrowing is always seen at the level of the esophageal hiatus of the diaphragm or about it, but never at the anatomic cardia. With the occluding mechanism between the esophagus and the stomach established at the level of the diaphragm, it seems reasonable to call the subdiaphragmatic structure the cardiac antrum of the stomach, rather than the abdominal portion of the esophagus. Embryologic data support this view, and roentgenologic observations conform with this new concept much better than with the traditional one. Whenever a subdiaphragmatic funnel-shaped structure can be visualized, the lining mucosa usually presents a rugal pattern similar to that of the stomach rather than to that of the esophagus.

The substitution of the term "functional cardia" for the old designation "anatomic cardia" is recommended and, as there is general agreement that the lowermost portion of the esophagus (called vestibule by some) has a sphincteric capacity over a length of 2 to 4 cm., it seems practical to call this the "internal sphincter" of the physiological cardia. The external sphincter is provided by the diaphragmatic muscle bundles that form the hiatus, the "pinchcock action" of the diaphragm.

The esophageal hiatus of the diaphragm is formed in man by fasciculi of the right crus. The esophagus is anchored to the rim of the hiatus by the phreno-esophageal ligament. This provides for rather wide lengthwise movements of the esophagus, and this flexibility is the main reason why it is so difficult to establish the topographic relations at the esophageal hiatus. In addition to the up-and-down movement of the esophagus as a whole, in relation to the diaphragm, the mucosal tube moves within the muscular tube of the esophagus. The transition from the normal condition to a hiatal hernia is gradual. A small hiatal hernia is frequently a physiological borderline situation without clinical significance; it may, however, have the inherent potentiality of becoming larger and significant.

Hiatal Hernia: The hiatus becomes wider with increasing age and variations in strength and tightness of the phreno-esophageal ligament may be assumed. Two types of hernia are distinguished: (1) those in which the esophagus and stomach have slipped through the hiatus as a whole in an axial fashion, known as axial or concentric hernia or, because of the incomplete peritoneal investment, as sliding hernia; (2) those in which the fundus of the stomach seems to have slipped upward along the esophagus, *i.e.*, paraesophageal or rolling hernia. Both types may be spontaneously reducible or permanently incarcerated. The paraesophageal hernias, except for their esophageal part, are usually completely covered by peritoneum. The author believes that the characteristic indentation between esophagus and herniated gastric pouch represents the persistent incisura cardiaca. In the axial type of hernia this indentation is effaced.

In both types of hernia a circular narrowing 2 cm. or more above the diaphragm is seen, representing the dislocated internal sphincter of the functional cardia. This weak sphincter, deprived by its dislocation of the support of the external sphincter, easily becomes incompetent, permitting reflux of gastric content into the esophagus.

Peptic Esophagitis: Peptic esophagitis is the source of most clinical symptoms associated with hiatus hernia. The welling up of gastric juice above the slipped cardiac sphincter can readily be demonstrated by esophagoscopy, and in the roentgenologic examination one may observe that barium flows back from the stomach into the esophagus. Filling of a herniated pouch in itself does not constitute abnormal reflux. Only backflow into the esophagus reveals incompetence of the sphincter and can be thus designated. A hiatus hernia with gastric juice reflux may not be constantly demonstrable. It may be seen only in certain positions or in the presence of muscular fatigue.

The severity of peptic esophagitis depends not only upon the strength of the gastric juice but on various body protective mechanisms, the lack of which is described as "ulcer diathesis." Superficial peptic esophagitis may not be visible roentgenologically. In more advanced cases mucosal swelling and ulceration become obvious. Severe esophagitis with extensive ulceration, deep fibrosis, and muscular hypertrophy, leads to stricture and lengthwise shrinking of the esophagus.

Mallory-Weiss Syndrome: This entity is characterized by esophageal lacerations associated with vomiting. The author lists the reasons why local sudden increases in intraluminal pressure may cause esophageal rupture, as with severe retching or vomiting, in the absence of any obstructing lesion in the proximal esophagus. The esophageal hiatus is usually wide when a hernia exists; thus, the force of the jet of the ejected gastric contents would not be broken at the hiatal level. The herniated gastric pouch is surrounded by low thoracic pressure rather than by strong intra-abdominal pressure. The slipped internal cardiac sphincter forms a strait above the hernia, thus adding to the building up of pressure within the hernia. Lastly, the slipped internal sphincter is a natural narrowing of the esophagus and, removed from its normally protecting hiatal ring, it is vulnerable to vehement, sudden distention. Some of the esophageal lacerations resulting from this mechanism may heal after some bleeding and need not necessarily be initially fatal. Thus the author concludes that the hiatus hernia is a prerequisite to the Mallory-Weiss syndrome.

Bleeding, Anemia, and Hiatal Hernia: Bleeding with hiatal hernia may be due to peptic esophagitis, Mallory-Weiss lacerations, or chronic marginal ulcer. Only in

the case of chronic marginal ulcer can the source of hemorrhage be directly seen roentgenologically. Esophagoscopy, however, may show a superficial esophagitis or lacerations. It is suggested that in some cases of severe esophagogastric hemorrhage which have been successfully treated by subtotal gastric resection, the reason for cure may well have been an unintentional repair of a hiatus hernia. After resection of the lower portion of the esophagus and esophago-gastrostomy, recurrent peptic ulcer and esophagitis are less frequent if a considerable portion of the stomach has been resected. Iron deficiency anemia, often of long duration and of marked degree, has been cured by repair of the hiatal hernia, probably because of the removal of a source of bleeding.

Esophageal or Marginal Ulcer: Marginal ulcers usually occur on the gastric side of the junction of the esophageal and gastric mucosa. It is suggested that such ulcers have originated from Mallory-Weiss lacerations rather than being due to islands of gastric mucosa in the lower esophagus or the result of autodigestive episodes on the gastric side of the esophagogastric junction.

Five roentgenograms; 2 photographs; 7 diagrams.

JOHN P. FOTOPOULOS, M.D.
Hartford, Conn.

THE DIGESTIVE SYSTEM

Some Observations Concerned with Digestive Tract Roentgenology. Parts I and II. Marlyn W. Miller. Pennsylvania M. J. **59:** 796–800, July 1956; 905–908, August 1956. (Altoona Hospital, Altoona, Penna.)

This article is a statistical review of one year's experience of a 300-bed general hospital with examinations of the digestive tract. Over a period of twenty years there had been a steady increase in such examinations to twelve times the number performed in 1934.

During the year 1953, 1,855 x-ray examinations of the digestive tract with contrast material were performed on 1,025 patients, about half of whom received two or more of the examinations at a single admission or visit. Of the 1,855 examinations, 1,515 were interpreted as showing no organic lesion. The total included 433 gallbladder studies, with 66 per cent regarded as normal; 712 gastrointestinal studies with the forward meal, with 76 per cent normal; 710 barium enema examinations of the colon, of which 95 per cent were considered as showing no organic intrinsic lesion. If, however, 118 diagnoses of spastic irritable colon are taken into consideration the percentage of entirely normal colon studies is decreased to 79.

The incidence of gastric or duodenal ulcer in the 712 gastrointestinal studies was 16 per cent; and 16 per cent of the large bowel examinations showed an irritable colon. The incidence of carcinoma of the digestive tract was 1.6 per cent.

Part II of this report presents some financial statistics. Payment for the 1,855 examinations was as follows:

Blue Cross insurance	48 per cent
Cash indemity	16 per cent
Full pay	15 per cent
Free	8 per cent

Sixty-four per cent of all examinations were covered by some form of hospital insurance requiring admission as a prerequisite to partial or complete payment for the x-ray examination. It is noteworthy that those having Blue Cross remained as in-patients the least number of average bed days.

Nine tables.

CADMAN CHAFFIN, M.D.
Mercy Hospital, Pittsburgh, Penna.

Clinical Observations on the Function of the Gastroesophageal Junction. Roy Cohn, Myron Close, and George Weston. Am. J. Surg. **92:** 194–200, August 1956. (San Francisco County Hospital, San Francisco, Calif.)

Marchand (Brit. J. Surg. **42:** 504, 1955) noted in cadavers that when water was pumped into the pyloric end of the stomach *in situ* in young adults, a pressure of 28 cm. of water was required to cause regurgitation into the esophagus. If the fundus were clamped off so that the stomach had the appearance of that seen in the "short esophagus," only 9 cm. of water pressure were required to cause regurgitation. If the diaphragm were removed so that the fundus could balloon into the thorax, thus increasing the gastroesophageal angle, 42 cm. of water pressure was necessary for regurgitation. Marchand interpreted these data to mean that the angle of the junction of the esophagus with the stomach was of importance in preventing reflux of gastric contents.

The present authors offer additional support of Marchand's thesis, obtained in living subjects. Ten patients being treated for tuberculosis by pneumoperitoneum and 21 with small esophageal hiatus hernias, were examined during the act of swallowing barium in upright and horizontal positions with and without pressure applied to the abdomen by a binder over an inflatable rubber bag.

In the upright position 4 of the pneumoperitoneum patients (all over fifty years of age) showed some regurgitation when pressure was applied but in the horizontal position there was no regurgitation with or without pressure. The authors ascribe the findings to the fact that the normal acute angle of the gastroesophageal junction was obliterated by the combination of the pneumoperitoneum and the pressure in the upright position, and was restored in the horizontal position. [Here it must be noted that the authors unfortunately seem to have used the terms prone and supine interchangeably, since illustrations called supine show the fundus of the stomach distended with barium and pressed against the diaphragm.]

In the hiatus hernia patients the incidence of regurgitation was higher, paralleling the degree to which the hernia obliterated the angle of the gastroesophageal junction.

It is concluded that the angle plays an important part in determining whether regurgitation will take place and that this should be taken into consideration in planning surgical procedures to prevent or control esophageal regurgitation and resultant stricture.

Fifteen roentgenograms.

ZAC F. ENDRESS, M.D.
Pontiac, Mich.

Cardio-Esophageal Relaxation (Chalasia) Studies on the Normal Infant. Leo Blank and Wilmer L. Pew. Am. J. Roentgenol. **76:** 540–550, September 1956. (University of Minnesota Hospitals, Minneapolis, Minn.)

After a review of the literature relative to chalasia, the authors report a series of 13 personal cases and describe a study undertaken to determine whether normal infants could be made to regurgitate a barium meal and,

if so, whether such regurgitation could be differentiated from chalasia. Examinations were carried out on 70 infants, four, five and six days of age, who were considered normal by physical examination and history relative to feeding and regurgitation. The swallowing function was observed fluoroscopically as the infant was fed a barium mixture through a nursing bottle. The usual thin mixture used for upper gastrointestinal studies was diluted with about an equal amount of the child's formula. Studies were made in the supine position and with the left side down in the oblique position. If regurgitation was noted, the infant was allowed to eructate swallowed air and further observations were made.

Thirty-two (46 per cent) of the 70 infants showed gastroesophageal reflux of some degree. During the phase of regurgitation, the cardia appeared patulous and the walls of the esophagus remained straight. In 31 cases peristalsis would force the barium back into the stomach. In the more severe cases reflux would recur as soon as the esophagus was emptied. In a single case there was free regurgitation. This was the only patient in whom poor esophageal tonus and weak peristalsis were observed. Re-examination two months later revealed no abnormalities in this infant.

The authors conclude that regurgitation can be seen as a normal phenomenon in a significant percentage of patients and does not establish the diagnosis of chalasia. For this there must be (1) a specific clinical picture, (2) certain roentgen findings, and (3) response to therapy.

1. Clinically there must be a history of persistent and excessive regurgitation beginning in the neonatal period. Signs of growth retardation, dehydration, aspiration pneumonia or esophagitis may be present as sequelae.

2. The roentgen findings reveal regurgitation of the barium mixture into the esophagus through a patulous cardia. The esophageal wall is flaccid with poor tonus and weakness or absence of peristalsis. A secondary peristaltic wave may start at the level of the arch of the aorta, but fades away, never compressing the lumen, nor traveling to the cardia. The esophagus is wider than usual and with moderately undulating walls. Congenital obstructive lesions in the duodenum or lower in the gastrointestinal tract may produce changes in the esophagus and cardia which cannot be differentiated from chalasia.

3. Response to thickened feedings and positioning is uniformly good. The authors suggest placing the child in the prone position with forty-five degree elevation of the head of the bed.

Speculation as to the cause of this condition is made, It is suggested that it is due to incomplete development of neuromuscular coordination.

Eight roentgenograms, 1 photograph, 2 tables.

JOHN P. FOTOPOULOS, M.D.
Hartford, Conn.

Gastro-Oesophageal Reflux in Infancy and Childhood. Frieda E. Plarre. M. J. Australia **2:** 241–246, Aug. 18, 1956. (Royal Children's Hospital, Melbourne, Australia)

Prior to the last ten years the phenomenon of gastroesophageal reflux was practically unknown. The author cites various contributions to its understanding published since that time, reviews briefly the radiological appearances of the esophagus in the normal infant, and reports her own observations in a series of 35 infants with cardio-esophageal relaxation or small hiatus hernia presenting symptoms during the first year of life. They were followed for varying periods ranging from one to five years and in all but one the course was benign. In this single exception, symptoms persisted and a large chronic peptic ulcer developed in the gastric hernia, which was excised at the age of six and a half-years. Eight case histories are presented.

The following conclusions are reached.

1. A hiatal defect may be present early in life without necessarily causing reflux.

2. A condition of simple cardioesophageal relaxation or of minor intrathoracic stomach may return to normal after the symptoms of reflux and gastroesophagitis have subsided if control is started early enough.

3. Reflux may persist long after the symptoms have subsided under medical control and also after surgical treatment in which adequate repair has been carried out.

4. The diagnosis of actual or impending fibrous stricture and its differentiation from narrowing of the esophagus due to spasm or edema is difficult in a young child. Radiological examination may be misleading, for narrowing due to muscle spasm or edematous infiltration of a submucosal tissue may persist with an unchanged picture for several weeks.

Clinical study shows no direct parallel between the degree of dysphagia and severity of a stricture, except in advanced cases, for dysphagia may occur with esophagitis and a wide atonic esophagus in the absence of any stricture, and alternately dysphagia may be masked in the case of a mild stricture by thorough mastication and slow eating habits. Careful correlation between clinical, radiological, and endoscopic findings must be combined with a reasonable period of close clinical observation in order to make an accurate diagnosis.

The pathological basis of the mucosal changes of the gastroesophageal region still requires clarification. Meanwhile, both endoscopic and radiological interpretation in the early stages of these clinical conditions must remain limited in scope.

Thirty-two roentgenograms; 4 diagrams.

ARTHUR JOHN COOK, M.D.
ATLANTA, GA.

A Double Contrast Method for the Roentgen Examination of Esophageal Strictures. Hans Freidenfelt. Acta radiol. **46:** 499–506, September 1956. (Karolinska Sjukhuset, Stockholm, Sweden)

With esophageal strictures, including those as the result of carcinoma and inflammatory disease, it is frequently difficult by ordinary esophageal examination to determine the extent of involvement, as the post-stenotic esophagus cannot be adequately distended. In approximately 100 cases the author has carried out examinations in the upright oblique position after the patient has ingested a relatively small amount of thin barium emulsion followed by a teaspoonful of equal parts of sodium bicarbonate and tartaric acid. The effervescent powder is washed down by a small swallow of the thin barium emulsion. Carbonic acid is thus formed, resulting in dilatation of the esophagus. Four and six exposures are made in rapid succession.

This method is applicable to all portions of the esophagus except for the hypopharynx, where it is contraindicated because of the risk of inhalation of the barium foam. The procedure can be repeated three or four times in the course of a single examination without dis-

comfort. The technic proved satisfactory in over 97 per cent of the author's cases. Its chief value lies in definition of the lower border of the stricture and, to some extent, in determination of the distensibility of the wall, information which is of value to the endoscopist attempting to pass an esophagoscope.

Seventeen roentgenograms. J. A. GUNN, M.D.
Grand Rapids, Mich.

Idiopathic Ulcerative Esophagitis. Report of a Case. Hart Achenbach, Joseph P. Lynch, and Richard W. Dwight. New England J. Med. **255:** 456–459, Sept. 6, 1956. (H. A., Boston City Hospital, Boston, Mass.)

The authors report an unusual case of idiopathic ulcerative esophagitis originating in the lower third and progressing over a period of months, eventually to involve the entire length of the esophagus. There were loss of mucous membrane and destruction of muscular coats in several places, leading to stenosis and multiple perforations, which caused abscesses in the mediastinum and right lung. Complicating processes included: perianal abscess and fistula, erythema nodosum, migratory arthritis, pyoderma gangraenosum, splenomegaly, and hepatic dysfunction.

The patient's first symptom was painless dysphagia. The first demonstrable abnormality was thickening of the longitudinal folds of the esophagus as seen on x-ray examination. The mucosa became fissured and then ulcerated, with undermining and pseudo-polyp formation. The esophagus lost its motility and became stenotic and obstructed.

Because of the complications present, the age of the patient (twenty-five years), and his rapid deterioration, a diagnosis of ulcerative colitis was entertained but was never substantiated by barium enema study or sigmoidoscopy. In any event, esophagectomy, performed as a life-saving procedure, apparently cured the associated arthritis and skin and perianal lesions, as colectomy does in ulcerative colitis. This appears to be a disease similiar to ulcerative colitis but occurring in the esophagus.

Eight roentgenograms.

THEODORE E. KEATS, M.D.
University of Missouri

Pulsion Diverticulum Simulating Foreign Body in Esophagus. Case Report. Albert R. Zavatsky. Pennsylvania M. J. **59:** 1075–1077, September 1956. (Pittsburgh, Penna.)

A case of hypopharyngeal diverticulum is reported, with signs and symptoms suggestive of a foreign body in the esophagus.

A 62-year-old white male complained of dysphagia, a sticking sensation in the upper neck, and a feeling of "something stuck in the throat." Soft-tissue roentgenograms of the neck in lateral projection demonstrated a small zone of translucency at the level of C-7, but no evidence of a foreign body, though the patient believed that he might have swallowed a chicken bone thirty hours prior to the onset of symptoms. Later studies with barium mixtures demonstrated a typical pulsion diverticulum of small size at the level of the cricopharyngeal muscle (C-6). Esophagoscopy showed inflammatory changes around the mouth of the diverticulum but no foreign body.

Four roentgenograms.

JAMES W. BARBER, M.D.
Cheyenne, Wyo.

Malignant Lymphoma of the Gastrointestinal Tract: Roentgenographic Considerations. C. C. Wang and James A. Petersen. Acta radiol. **46:** 523–532, September 1956. (Massachusetts General Hospital, Boston, Mass.)

In approximately 10 per cent of lymphoma patients the gastrointestinal tract is involved. The authors studied 165 such cases. In 89 of these the disease appeared to arise primarily within the gastrointestinal tract, while in the remainder the lesion was part of a generalized process. The stomach is most commonly involved followed by the small bowel. Lymphoma of the esophagus and colon is rare.

In the present series the stomach was involved in 82 cases. Frequently gastric lymphoma cannot be differentiated from carcinoma. The presence of giant rugal folds involving a large portion of the stomach, extensive involvement but little impairment of peristalsis, multiple gastric ulcers, and a large gastric ulcer and/or palpable tumor, particularly in a young patient, are all considered strongly suggestive of lymphoma. If the patient has generalized lymphoma, the diagnosis is obviously easier.

Small bowel lymphoma occurred in 59 of the authors' patients. It most commonly took the form of multiple ulcerating lesions separated by areas of relatively normal appearing mucosa. There was some associated widening of the intestinal lumen, and the involvement varied in length. Occasionally a large soft-tissue mass was seen, traversed by irregular air-containing channels which, on barium study, were shown to be the lumen of the involved bowel. The mucosa was usually destroyed or replaced by tumor. Again, slight dilatation of the lumen and fixation were encountered. Nodular lesions 1 to 2 cm. in size were found in 1 case. Another patient had diffuse involvement of the mucosa, mimicking the so-called "deficiency pattern."

The diagnosis of lymphoma of the intestinal tract is not difficult if it is part of a generalized process. Primary lesions can seldom be diagnosed conclusively without a biopsy, although some of the changes described above are sufficiently reliable to suggest lymphoma rather than carcinoma, benign ulcer, sarcoma, or a retroperitoneal tumor.

Eleven roentgenograms. J. A. GUNN, M.D.
Grand Rapids, Mich.

Ulcers Along the Greater Curvature of the Stomach; Observations on Three Cases. K. Axmann and J. Šetka. Fortschr. a. d. Geb. d. Röntgenstrahlen **85:** 303–309, September 1956. (In German) (K. A., Rooseveltplatz 5, Tábor, Czechoslovakia)

Benign ulcers of the greater curvature of the stomach are relatively rare. In the English literature all ulcers of the greater curvature are considered to be potentially malignant, and surgical treatment is recommended. The French, on the other hand, often treat these ulcers conservatively, apparently with good results.

Ulcers of the greater curvature are often overlooked, possibly on account of the coarse rugal pattern in this area. The diagnosis should be based on the demonstration of a "niche." In this area, however, the movements of the mucosa are more free than in the relatively fixed lesser curvature, and the mucosal folds may creep over the base of the ulcer, producing in a tangential projection a "meniscus appearance" which may lead to the erroneous diagnosis of carcinoma. If the ulcer is in the vertical portion of the stomach, there is often an

oblique mucosal fold leading up to it, which has some differential diagnostic significance in that it is not found in carcinoma. Some authors believe that ulcers in the vertical portion of the stomach are for the most part benign, while those of the horizontal portion are always malignant.

Diverticula can be differentiated from ulcers by the smooth, regular outline, the presence of mucosal folds within the neck and the cavity of the diverticulum, and by the fact that they can be freely moved around, while ulcers with niches are usually fixed, attached to the surrounding structures, and without movement on respiration.

The authors present 3 proved cases of multiple ulcers of the greater curvature, in which the diagnosis was made before operation, by x-ray and gastroscopic examination. Their conclusion is that the old dictum that "an ulcer of the greater curvature is a carcinoma" is incorrect.

Nine roentgenograms; 3 photographs.

WILLIAM A. MARSHALL, M.D.
Chicago, Ill.

Motility of the Small Intestine with Non-Flocculating Medium; a Review of 173 Roentgen Examinations. Israel E. Kirsh. Gastroenterology **31**: 251–259, September 1956. (Department of Radiology, Veterans Administration Hospital, Hines, Ill.)

The author studied transit time in the small intestine in 173 patients without known small intestinal disease. A "non-flocculating" medium, consisting of a stable suspension of barium sulfate in water containing 0.25 per cent of sodium carboxymethylcellulose, was used.

Approximately 18 per cent of all patients had a transit time of one hour or less; in 8 the transit time was only fifteen minutes. Another 18 per cent had a transit time of five hours or longer. In 50 per cent of the series the time was between one and one-half and three hours. The median time was two hours. When multiple studies were done on one patient with an interval of approximately one week, the transit time was found to be fairly constant.

The author agrees with statements made previously that the transit time is usually somewhat shorter with a nonflocculating medium than with ordinary barium sulfate.

The conclusion is reached that there is so much normal variability in motility that the term "hypermotility of the small intestine" is practically meaningless.

Two roentgenograms; 3 tables.

RICHARD A. ELMER, M.D.
Atlanta, Ga.

Volvulus in Western India. A Clinical Study of 40 Cases, with Particular Reference to the Conservative Treatment of Pelvic Colon Volvulus. D. A. Andersen. Brit. J. Surg. **44**: 130–143, September 1956. (Evangeline Booth Hospital, Ahmednager, India)

The author's experience in Western India for fifteen years indicates that volvulus, both of the small and large intestine, is common there. During the years 1948–53, 168 cases of acute or subacute intestinal obstruction were treated in the mission hospital from which this report comes, representing 2.5 per cent of surgical admissions, compared with 0.85 per cent in Massachusetts General Hospital (quoted by Wangensteen in *Intestinal Obstruction*, 2nd ed., 1949). Volvulus was the cause of obstruction in 40 of the 168 cases, or 24 per cent, as compared to 4 per cent in Wangensteen's series.

Three major factors are considered as predisposing causes of volvulus: (1) inflammatory adhesions; (2) congenital anomalies, such as peritoneal bands and mobile cecum; (3) diet, suggested by observations on vegetarians.

Volvulus of the pelvic colon may be "benign" or "serious," depending upon the presence or absence of symptoms suggestive of gangrene. In the benign type the clinical picture was as follows: sudden onset of severe abdominal pain associated with vomiting for two to five days, constipation, and increasing abdominal distention. Inspection showed the distention to be extreme, more marked on the left and in the upper abdomen. The general condition of these patients and the pulse (rate 80–90) were surprisingly good. The radiological findings on the plain upright radiographs included (1) a grossly distended double pelvic loop with fluid levels, (2) a variable amount of gas and fluid in the remainder of the colon, and (3) occasionally a few fluid levels in the lower small bowel.

Five of the author's cases were of the serious type, with severe pain, marked diffuse tenderness, a silent abdomen, and a weak and rapid pulse. Three of these patients were first seen in extreme surgical shock and on operation total gangrene of the pelvic loop was found.

For the benign type of volvulus of the pelvic colon, a conservative reduction at the time of the attack is recommended, followed after seven to ten days by a one-stage resection. In the presence of gangrene, the simplest treatment is exteriorization of the gangrenous loop followed by a Paul-Mikulicz resection. This, however, entails a definite risk of gangrene of the lower stump due to thrombosis of the sigmoid branches of the inferior mesenteric artery. The author therefore closes both ends of the pelvic loop after resection, making sure that this is carried out through well nourished bowel wall, with a proximal colostomy, preferably of the right transverse colon.

Volvulus of the small intestine was present in 14 cases of this series. The duration of symptoms prior to admission varied between twenty hours and twelve days. There was a severe constant pain in most cases; in others, a colicky pain was present. Vomiting was often persistent. The pulse was 100 or higher. The patients were usually afebrile. The abdomen was moderately distended, and in several instances definite peristalsis was visible, associated with high-pitched sounds on auscultation. A plain radiograph with the patient standing gave definite evidence of small intestinal obstruction, with inverted distended loops showing fluid levels. A distinction between volvulus and other obstructions could not be made preoperatively. The treatment was laparotomy with simple untwisting in 9 cases, suture of the local gangrene line in 2 cases, and resection in 3 cases.

Six roentgenograms: 15 diagrams; 1 graph; 4 tables.

JOACHIM GFOELLER, M.D.
Cleveland City Hospital

Volvulus of the Splenic Flexure. Maxwell H. Poppel, Bertram E. Zeitel, and Raymond M. Abrams. Am. J. Digest. Dis. (n.s.): **1**: 380–386, September 1956. (New York University-Bellevue Medical Center, New York, N. Y.)

Two proved cases of volvulus of the splenic flexure of

the colon are added to the 1 operative case reported in the literature (Buenger: Am J. Roentgenol. **71:** 81, 1954). Both patients were elderly men with a history of pain for about a week, abdominal distention, and decreased bowel movements. A barium enema study in each case showed marked dilatation and twisting of the splenic flexure, confirmed by operation. The first patient, who gave a history of previous episodes of pain and distention, had agenesis of the gastrocolic ligament and lateral peritoneal-colic attachments. The second patient had a short mesocolon at the splenic flexure and a benign stricture of the sigmoid which apparently predisposed to distention and twisting of the splenic flexure. Both patients were successfully treated by partial colectomy.

Five roentgenograms. CAPT. GARTH R. DREWRY
Tampa, Fla.

Spontaneous Regression of Polyps of the Colon in Children. Lars Andrén and Sture Frieberg. Acta radiol. **46:** 507–510, September 1956. (Malmö Allmänna Sjukhus, Malmö, Sweden)

Polyps of the colon are especially common in children in the four- to six-year-old age group. Their frequency then decreases, only to increase again in adult life. The fact that the polyps diminish in number or disappear during the interval indicates that they either spontaneously drop off from the colon or regress.

Two cases of polyps visualized in five-year-old patients are reported. Without operative intervention, repeat studies eighteen to twenty months later showed considerable regression in the size of the tumors. These 2 cases afford further evidence of the spontaneous regression of polyps of the colon in childhood and indicate that surgical intervention in these cases may be unnecessary.

Four roentgenograms. J. A. GUNN, M.D.
Grand Rapids, Mich.

The Emptying Rhythm of the Normal Gallbladder Visualized with Tri-Iodophenylpropionic Acid (Telepaque). Constant Wieser. Radiol. clin. **25:** 287–296, September 1956. (In German) (Zentral Röntgeninstitut des Kantonsspitals St. Gallen, Saint Gallen, Switzerland)

With the aid of a previously developed method of measuring quantitatively the volume of the gallbladder (Schweiz. med. Wchnschr. **84:** 149, 1954. Abst. in Radiology **63:** 774, 1954), contraction of the healthy gallbladder following administration of Telepaque was studied in 27 persons. There was no essential difference between the male and the female. The effect of the fatty meal was found to be maximal at twenty to thirty minutes, indicating the most favorable time for making the exposures to detect the contracting ability of the gallbladder. Incidentally, at this time the large bile passages were visualized in three-quarters of the patients. The occasional patient will even show an increase in volume of the gallbladder at ten minutes. This is temporary and is considered to be due to the choleretic action of egg yolk.

The author's material is formulated statistically, with the arithmetic mean of percentage contraction following a fatty meal, and one and two standard deviations above and below this mean.

Eight roentgenograms; 7 drawings; 1 table.
CHRISTIAN V. CIMMINO, M.D.
Fredericksburg, Va.

Benign Tumors of the Gall Bladder. Seymour Ochsner and G. M. Carrera. Gastroenterology **31:** 266–273, September 1956. (Ochsner Foundation Hospital, New Orleans, La.)

The authors point out that there is wide disagreement about the incidence of papilloma of the gallbladder, apparently due to the diversity of terminology and concept as to what constitutes a true papilloma. Some reports fail to differentiate between an actual neoplasm and a hypertrophied villus containing cholesterol (usually called cholesterol polyp or cholesterol papilloma).

The pathologic and clinical aspects of benign gallbladder tumors are discussed. In the authors' series of 10 cases there was 1 in which carcinoma *in situ* was demonstrated. Clinically, the most important aspect is generally considered to be the absence of symptoms. Stones may co-exist, as in 5 of the authors' cases.

A list of roentgen criteria for diagnosis is included. The condition may be supported by a fixed filling defect, usually single and frequently in a gallbladder which is otherwise normal in appearance.

The authors mention 2 cases (not included in this series) in which a mucosal polyp and a cholesterol polyp were mistakenly diagnosed as papilloma or adenoma.

[The authors correctly point out that the confusion with respect to benign tumors of the gallbladder is at least in large measure due to varied terminology. They indicate that a sharp differentiation must be made between tumors which are truly neoplastic and those which are not. In their discussion of the pathologic aspects, however, they fail to straighten out the variation in terminology. It would seem to the abstracter to be wise, at least until such time as the nomenclature is reasonably well standardized, to qualify the term papilloma or polyp of the gallbladder with the additional term "inflammatory type" or "true neoplasm," as the case may be. Confusion will exist so long as descriptive and pathogenetic terms are used interchangeably—R.A.E.]

Six roentgenograms; 2 photomicrographs.
RICHARD A. ELMER, M.D.
Atlanta, Ga.

"Polyposis" of the Gall-Bladder. D. J. Oakland. Brit. J. Surg. **44:** 203–206, September 1956. (Birmingham, England)

Two cases of polyposis of the gallbladder are described which, though similar radiologically and macroscopically, represent two types of polyp. In each case a cholecystogram showed several negative shadows which appeared to arise from the wall of the gallbladder.

In *Case I* there was a history of chronic cholecystitis. Cholecystectomy was performed, and examination of the gallbladder revealed many small polyps, 4 to 10 mm. in height. Histologically the mucosa showed diffuse papillomatous hyperplasia. The papillomata were covered with columnar epithelium, beneath which was a fibrovascular stroma heavily infiltrated with lipid-containing histiocytes ("foam cells"). The submucosa showed chronic inflammatory cells.

In *Case II*, the gallbladder appeared normal on inspection, but palpation revealed several nodules. Cholecystectomy was performed. The specimen showed multiple small polyps without other evidence of disease. Histologically each papilloma consisted of a complex mass of fronds lined by columnar epithelium, which covered a sparse fibrovascular stroma. Slight chronic inflammatory cell infiltration in the stroma of

the papillomata and in the uninvolved gallbladder wall was present.

Phillips (Am. J. Surg. **21**: 38, 1933) reviewed 500 cases of papilloma of the gallbladder seen at the Mayo Clinic and distinguished two varieties: (1) the inflammatory type, (2) the inflammatory and metabolic type. As Tabah and McNeer (Surgery **34**: 57, 1953. Abst. in Radiology **62**: 779, 1954) point out, however, neither type is strictly neoplastic. The first of the cases described above corresponds to the inflammatory and metabolic type. The second case is a true papilloma.

Two roentgenograms; 3 photomicrographs; 1 photograph.

JOACHIM GFOELLER, M.D.
Cleveland City Hospital

Action of Bile Acids on Clearance of Biligrafin from the Plasma. Is Biligrafin a Model of Bilirubin Metabolism? Preliminary Report. Istvan Lajos. Fortschr. a. d. Geb. d. Röntgenstrahlen **85**: 299–302, September 1956. (In German) (II. Klinik für Innere Medizin der Medizinischen Universität Szeged, Szeged, Hungary)

Rabbits weighing 4 to 5 kg. were given 1 gm. of Biligrafin (Cholografin) intravenously. X-ray films showed an intense shadow of the gallbladder. The same rabbits, a few days later, were again given Biligrafin, but this time with 5 ml. of 20 per cent Decholin. No gallbladder shadow was now obtained. A similar observation was made on 2 healthy human beings. Systematic examinations of the iodine level of the blood serum showed that, one hour after the injection, the iodine content was many times higher in the animals that had received the Decholin. Decholin was thus shown to delay the excretion through the liver.

Further experiments indicated that the non-visualization of the gallbladder after Decholin is due not to changes in the blood serum but rather to the fact that the presence of bile acids (Decholin) in the blood produces a change in the permeability of the liver cells. The Biligrafin, instead of entering the bile system of the liver, is re-routed back into the blood stream. This is similar to the re-routing of urobilinogen in cases of liver damage. An alteration of the permeability of the liver cells prevents the absorption of urobilinogen by the liver from the blood stream; the level in the blood rises, and excretion eventually occurs through the kidneys. In analogy a delay in the excretion of Biligrafin by the liver may be expected if liver damage exists and if urobilinogen appears in the urine.

Biligrafin, when injected into the blood, attaches itself for the most part to the serum proteins. About 20 per cent remains unattached and this is mainly excreted by the kidneys, while that bound to serum albumin is excreted through the biliary system. In this respect Biligrafin metabolism is very similar to bilirubin metabolism.

One roentgenogram; 6 graphs; 2 tables.

WILLIAM A. MARSHALL, M.D.
Chicago, Ill.

THE MUSCULOSKELETAL SYSTEM

Roentgenographic Investigation of the Human Skeleton During Early Fetal Life. Ronan O'Rahilly and David B. Meyer. Am. J. Roentgenol. **76**: 455–468, September 1956. (Wayne State University College of Medicine, Detroit, Mich.)

The authors studied 77 fetuses of undetermined sex, ranging from 49 to 150 mm. in crown-rump length, to determine the time of initial appearance of the ossific centers in the skeleton, and to obtain information as to their subsequent prenatal growth and fusion.

The fetuses studied were fixed in 10 per cent formalin, then placed in a 0.5 per cent aqueous solution of silver nitrate. Roentgenograms were made before and after the silver impregnation. The observations are presented in four tables and two graphic charts as well as in the text. The original article should be consulted for detailed information.

The authors consider the silver roentgenographic technic a satisfactory one for the study of osseous tissue in the period of fetal life covered by their investigations. The results do not differ significantly from those with alizarin staining, and the method is simpler and less time-consuming. It allows of subsequent histologic studies.

Twelve roentgenograms; 3 photomicrographs; 3 charts; 2 diagrams; 4 tables.

WILLIAM S. HARWELL, M.D.
Confederate Memorial Hospital, Shreveport, La.

Chemical Growth Dynamics of the Skeleton in the Immature Rat. III. Correlation of Growth and Morphology of Long Bones with Chemical Growth in Normal and Vitamin D-Deficient Animals. D. E. Pickering, L. B. Lusted, R. F. Foran, and J. T. Crane. J. Dis. Child. **92**: 292–296, September 1956. (D. E. P., University of Oregon Medical School, Portland, Ore.)

In a well controlled experiment the authors studied the chemical growth of the skeleton in immature male Wistar rats. One group was maintained on a normal diet and another was given a diet deficient in vitamin D. The animals were killed at one, twenty-one, forty-five, and seventy-five days of age. Despite the achievement of an overall reduction in skeletal calcium in the vitamin-D deficient rats, there was no gross or roentgenographic difference in the appearance of the tibias as compared to those of normal rats at forty-five and seventy-five days. At seventy-five days there were microscopic differences consisting of an increase in the thickness and cellularity of the epiphyseal cartilage with irregularity of the usually columnar arrangement of the cartilage cells. No significant microscopic alterations of the individual trabecular architecture, bone resorption, or remodeling were noted.

It would appear that, while the deficient diet produced significant changes in chemical growth, this was not mirrored by a change in linear growth of the tibia. [In the reproductions of tibial roentgenograms from the two groups of rats there appear to be differences in mineralization, metaphyseal flaring, and distal epiphyseal fragmentation.—S.S.]

Two roentgenograms; 2 photomicrographs; 2 graphs; 1 table.

SAUL SCHEFF, M.D.
Boston, Mass.

Arterial Supply to the Head of the Femur. An Arteriographic Study in Vivo of Lesions Attending Fracture of the Femoral Neck. Herbert Müssbichler. Acta radiol. **46**: 533–546, September 1956. (Lasarettet, Falun, Sweden)

This paper presents a study of the vessels of most importance in the nutrition of the femoral head by arteriography *in vivo*. In the majority of the cases the femoral artery was punctured percutaneously at the level of the inguinal ligament and 15 to 20 c.c. of Dijodone

50 per cent was injected. In some instances the deep femoral artery was punctured instead of the femoral, and in some cases a catheter was introduced into the artery. Four exposures were made in five to six seconds, beginning during the injection of the last 5 c.c. of the contrast material.

A total of 28 diseased hips in 25 patients were examined. This series included 4 cases of necrosis of the femoral head, 2 of pseudarthrosis, 1 of carcinomatous metastases in the acetabulum, 2 of known and 2 of suspected Perthes' disease, 2 of dislocation, 1 of Judet's plastic operation, as well as 3 pertrochanteric and 11 medial fractures of the femoral neck. Examination of the contralateral hip was done in 19 cases.

The main purpose was to study the posterior collum branch of the medial circumflex artery and the inferior and superior synovial arteries, in view of their size and importance.

On examining normal hips (15 cases), the following arteriographic features were invariably present:

1. Contrast-filling of the medial and lateral circumflex arteries of practically equal density, irrespective of variations in origin and course.
2. Contrast-filling of the posterior collum branch, its course being visualized to the intertrochanteric notch.

The inferior synovial artery was contrast-filled in 13 cases.

Study of the affected hips failed to show that the general contrast filling of vessels on the diseased side was less marked than in the normal hip. On the contrary, the anastomoses between the different arterial systems were in several cases of fracture of the femoral neck strikingly well filled. Some local changes, however, were demonstrable in the posterior collum branch: dilatation, decreased rate of flow, and arrested circulation. Since similar changes were not observed in the normal hips, they are taken to be of pathologic significance. Five short case reports are included, with radiographs demonstrating these features.

Seventeen roentgenograms.

J. P. CHAMPION, M.D.
Grand Rapids, Mich.

Radiological Study on the Trabeculae of the Humerus. Yoshinori Nakamura. J. Radiol. & Phys. Therapy, Univ. Kanazawa **36:** 181–211, September 1956. (In Japanese, with English abstract) (Department of Radiology, Faculty of Medicine, Kanazawa University, Kanazawa, Japan)

Frontal views of the proximal end of the humerus reveal trabeculations which can be classified by their direction into six "systems" of lines, which the author numbers from H-1 to H-6; these "systems" are also identifiable on lateral views, which show still another system, H-7. Frontal views of the distal humeral end show six similar systems of trabeculae, designated Y-1 through Y-6, but on lateral views there is too much overlapping for adequate evaluation. The upper (H) and lower (Y) systems intertwine in the diaphysis.

There is a definite relationship between muscular strength and trabeculation, especially system H-1 (extending between the greater tuberosity and the medial cortex of the proximal third of the shaft). The trabeculae constitute the lines of force along which the bone provides structural support for muscular contraction.

Studies performed in 122 older persons revealed noticeable changes in the humeral trabeculation occurring around the age of forty, although individual differences were of significance. Osteoporosis (senile atrophy) played a major role, as well as the decrease in muscular strength (pull), which was most evident about the humeral tuberosity.

Following an injury, arrangement of the trabeculae may be modified according to the site of the fracture and/or dislocation, subsequent functional disturbances, and the individual's muscular strength. When the H systems are fractured, there is generally a tendency toward varus deformity; with fracture of the Y systems the tendency is toward humerus valgus. During callus formation, there is thickening of the trabeculae, but restoration of the original appearance is often achieved when healing is complete.

Six roentgenograms, 31 drawings, 2 tables.

HYO HYUN BYUN, M.D.
Cook County Hospital, Chicago

The Ischiopubic Synchondrosis in Healthy Children: Some Normal Roentgenologic Findings. John Caffey and Steven E. Ross. Am. J. Roentgenol. **76:** 488–494, September 1956. (Babies Hospital, New York 32, N. Y.)

The material for this study consisted of 549 roentgenograms of the pelvis of normal children between the ages of two and twelve years. It was found that fusion of the ischiopubic synchondrosis takes place at widely differing intervals in normal children. While complete bilateral fusion of this joint had occurred in 6 per cent of the children by the age of four years, in 17 per cent fusion was still not complete at the age of twelve. The previously considered normal fusion time of four to eight years is therefore not accurate and the normal period must be extended to twelve years or beyond.

Bony swelling and uneven mineralization at the synchondrosis were present between the age of six and ten years in more than half the children. Since this must be considered a normal finding, ischiopubic osteochondritis (Van Neck's disease), which has been diagnosed on the basis of similar roentgenographic findings, must be regarded as "exceedingly rare."

Twenty roentgenograms; 2 tables.

RICHARD G. LESTER, M.D.
University of Minnesota

Ischiopubic Osteochondrosis, Van Neck and Odelberg's Disease. G. Lathion and J.-P. Christen. Radiol. clin. **25:** 282–287, September 1956. (In French) (Institut universitaire de radiologie de Lausanne, Lausanne, Switzerland)

While the authors' main conclusion is well known, namely, that the diagnosis of ischiopubic osteochondrosis cannot be made solely on the roentgen findings, this paper was considered worthy of an abstract because of the thorough manner in which this point is proved.

Ischiopubic osteochondrosis is found at the line of closure of the ischiopubic joint, between six and ten years of age in the majority of cases. As in other aseptic necroses, the cause is unknown. The patient complains of pains of varying intensity localized not only in the pubis but also in the hip, the internal surface of the thigh, and in the knee. A limp may appear with fatigue. The pains and limp are intermittent. Objectively, a slight functional limitation of the hip is noted. Rectal examination reveals tenderness in the ischiopubic synchondrosis and at times a hard tumefaction at this level. The laboratory study is unimportant, except to exclude tuberculosis.

Radiologically, widening and a microgeodic appearance are found in the synchondrosis. Involvement may be either unilateral or bilateral.

Films from 270 children of five to twelve years, hospitalized for unrelated conditions, were examined with particular reference to the ischiopubic synchondrosis. The appearances of this joint could be divided into six groups. In the first three of these six groups there was no suggestion of abnormality: the synchondrosis was smooth and not ballooned. The other three groups showed some cause for suspicion: the synchondrosis was somewhat dilated, with or without multiple rounded rarefactions. Had the patients in these last three groups had symptoms, a diagnosis of ischiopubic osteochondrosis would have been made. This appearance, however, was seen especially in patients who had diseases of the hip, such as congenital subluxation and aseptic necrosis. This would suggest that simple static difficulties might be associated with this peculiar appearance in the ischiopubic synchondrosis, which, when associated with symptoms, would be called aseptic necrosis.

Two roentgenograms; 1 line drawing.

CHRISTIAN V. CIMMINO, M.D.
Fredericksburg, Va.

Calcifying Disk Disease. S. de Sèze, A. Djian, and R. Claisse. Rev. du rhum. **23:** 1–17, April 1956. (In French) (Institut National d' Hygiène, Paris, France)

The case of a 7-year-old boy with torticollis and pain in the neck, impeding movement, is reported. The child had frequent episodes of sore throat, the last such episode, one month before the appearance of the torticollis, having been accompanied by fever reaching 104° F. Examination revealed stiffness of the neck with pain and tenderness. Roentgenograms showed calcification in the central part of the disk space between C-6 and C-7.

The pain, tenderness, and stiffness had disappeared when the child was again examined fifteen days later, and films of the cervical spine obtained after a lapse of a year and a half showed complete disappearance of the calcification. In reviewing the literature the authors found 8 similar cases.

Disk calcification in children is of a different nature than in adults. In childhood the disk is supplied by several vessels and thus is accessible to infection by the vascular route. Calcifications of infectious etiology are associated with febrile episodes and evidence of infection elsewhere. The origin of the pain is not well understood.

(See Peacher and Storrs: Cervical Disk Calcification in Childhood. Radiology **67:** 396, 1956. Ed.)

Twelve roentgenograms.

ALEXANDER R. MARGULIS, M.D.
University of Minnesota

Pathological Fractures. Treatment by Internal Fixation and Irradiation. M. B. Devas, J. W. Dickson, and A. M. Jelliffe. Lancet **2:** 484–487, Sept. 8, 1956. (A.M.J., Middlesex Hospital, London, England)

The authors' results with internal fixation in a series of 18 pathological fractures (8 of the shaft of the femur, 7 of the upper femur, 3 of the shaft of the humerus) have been very satisfactory. In all cases there was considerable or complete relief of pain, and some patients with widespread malignant disease were able to resume an active life. Postoperative pain was easily controlled, and no patient died as a result of the procedure.

The indications for internal fixation of fractures through metastatic tumor deposits are: (1) when the fracture is painful or immobilizes the patient; (2) when external fixation is not as efficacious or does not allow early ambulation; (3) when the malignancy of the tumor is such that widespread deposits can be assumed to be already present, although they are not clinically apparent; (4) when deposits are known to exist elsewhere; (5) when the tumor is likely to be hormone-dependent.

The authors believe that the management of all pathological fractures through metastatic deposits should include radiotherapy. Even if the tumor is thought to be relatively radioresistant, a high tumor dose can be delivered through multiple ports around the limb circumference. This would be difficult if the fracture were concealed behind the appliances necessary for immobilization by conventional methods. Unless internal fixation is undertaken prophylactically or for the immobilization of a solitary metastasis of extremely low-grade malignancy, the irradiation should be administered postoperatively. Delaying the reduction of the fracture will reduce the chance of a satisfactory mechanical result, and the danger of disseminating the tumor by operating before irradiation need not be weighed very seriously in the type of case that is usually considered for the procedure. Irradiation fields should cover not only the fracture site but the whole of the affected bone, since malignant cells will be carried in with the nail.

The presence of a metal pin in the treatment field is associated with certain theoretical disadvantages. With 250 kv and a half-value layer of about 1–2 mm. Cu, some shadowing will occur behind the pin, owing to increased differential absorption in this metal. This can be largely obviated by the use of two or more opposing fields. In any event, the aim is not necessarily the complete destruction of every malignant cell in the affected area, but palliation before new metastases give rise to further symptoms.

The dose required depends on the radiosensitivity of the tumor. If histologic examination shows it to be anaplastic or having its origin from a primary growth that is radiosensitive, 2,000 to 3,000 r over one to two weeks will usually suffice. If the metastasis is from a well differentiated radioresistant tumor, a higher dose may be necessary, delivered over a correspondingly greater time. If the metastasis is from a hormone-dependent tumor, additional benefit may be obtained by the administration of testosterone and stilbestrol or by hormone surgery. These measures will not only help the local fracture to heal, but may also postpone the appearance of metastases elsewhere.

Six roentgenograms.

Alkaptonuria with Ochronosis and Arthritis. E. A. Rodko. J. Canad. A. Radiologists **7:** 29–32, September 1956. (Vancouver, B. C.)

A 52-year-old white male gave a long history of intermittent and progressive pain and stiffness in the large joints of the body, most marked in the hips and knees. Darkening of the skin and the sclerae had been present for seventeen years. The cartilages of the ears were thickened, hardened, and partly calcified. The lumbodorsal spine was rigid but not painful. Consid-

erable limitation of motion was shown in both hips and knees, especially on the left. Over a three year follow-up period the arthritis gradually increased, progressing to severe limitation or fusion of the involved joints. A cup arthroplasty of the right hip was performed, with marked improvement in position and movement. Biopsy at this operation confirmed the diagnosis of alkaptonuria with ochronosis and degenerative osteoarthritic changes.

A review of the literature shows alkaptonuria associated with arthritis and ochronosis to be of rare occurrence. In the earlier stages, the radiologic findings mimic both atrophic and hypertrophic or degenerative arthritis. As the disease progresses, the degenerative features become more pronounced, with bone fusion as the last stage.

Nine roentgenograms; 6 photographs.

JAMES W. BARBER, M.D.
Cheyenne, Wyo.

THE SPINAL CORD

Meningeal Diverticula of Sacral Nerve Roots (Perineurial Cysts). Kenneth J. Strully. J. A. M. A. **161:** 1147–1152, July 21, 1956. (20 Fifth Ave., New York 11, N. Y.)

The author emphasizes the importance of x-ray signs in establishing a preoperative diagnosis of meningeal diverticula of the sacral nerve roots (so-called perineurial cysts). These lesions are believed to be congenital in origin but may not manifest themselves until the fourth to the sixth decade of life. The lesions consist in cyst-like diverticula of the nerve sheath with microscopic connection with the subarachnoid space. They produce pain by compression and stretching of nerve fibers. No motor, sensory, or reflex changes are observed. The diagnosis should be considered in patients with non-specific root pain when other causes have been excluded.

Roentgen findings, previously disregarded, are summarized as follows: "1. Intrasacral lesions, either meningoceles or root diverticula, produce pressure atrophy in the sacrum. This has been seen with careful x-ray examination of the sacral canal in the lateral projection. The anteroposterior views may show erosion of the body and dorsal plate or ballooning of the lateral cortex of the sacrum. 2. Asymmetry or deformity of the caudal end of the subarachnoid space, seen in myelograms, is strongly suggestive of a mass lesion with the sacrum. 3. The free flow of iophendylate into large saccular dilatations within the sacral canal or prolongation of the caudal sac below the upper one-fourth of the sacrum strongly suggests a congenital deformity and is frequently associated with diverticula of the sacral root sleeves. 4. Globular collections of radiopaque oil within the sacral canal, separated from the normal subarachnoid space, appearing hours, days, or weeks after myelography, and associated with focal atrophy of the sacrum, are indicative of diverticula of root sheaths."

The author adds 4 cases with positive roentgen findings to 1 previously reported in an Addendum to a series of "lumbar and sacral cysts of meningeal origin" (Strully and Heiser: Radiology **62:** 545, April 1954). The operative findings in each instance confirmed the diagnosis.

Seven roentgenograms.

ARTHUR S. SHUFRO, M.D.
University of Michigan

GYNECOLOGY AND OBSTETRICS

Roentgen Determination of Length of Fetus in Utero. M. Worm. Fortschr. a. d. Geb. d. Röntgenstrahlen **85:** 320–325, September 1956. (In German) (Diabetikerheim, Kreis Greifswald, Karlsburg, Germany)

It has been customary in the author's clinic to induce labor in pregnant diabetics providing the fetus has obtained a sufficient degree of maturity. A well known criterion of maturity is the appearance of the epiphysis at the distal end of the femur, which, according to Christie, should be present by the thirty-sixth to the thirty-eighth week of gestation. However, since this epiphysis often cannot be seen because of technical and other difficulties, the author has resorted to direct measurement and computation of the size of the fetus from the x-ray film according to the methods of Wegrad (Zentrabl. f. Gynäk. **61:** 373, 1937). Maturity is considered to be present if the fetus is at least 48 cm. long.

The technic is as follows: On a film obtained with the patient prone (focal spot-film distance, 125 cm., 90–95 kv; mas according to the thickness of the patient), a thin lead wire is placed along the outer edge of the vertebral bodies from the distal edge of the fifth sacral to the proximal edge of the first cervical vertebra. The wire is then straightened out and its length is measured in centimeters. The true length of the spine is obtained according to the following formula:

$$\frac{\text{True length of spine}}{\text{Measured length of spine (from film)}} = \frac{\text{Focus-film distance} - (\frac{1}{2}\text{ patient thickness} + \text{distance between table top and Bucky})}{\text{Focus-film distance}}$$

True length of the spine thus obtained is multiplied by the constant coefficient 2.29 (according to Wegrad), which then gives the computed length of the fetus.

The author presents 64 cases in which the length of the fetus thus computed is compared with the actual length as measured after delivery. The margin of error is plainly illustrated on a graph. In half of the cases the error was less than 0.5 cm. In only 3 instances did the error exceed 4.5 cm., and never was it greater than 5.5 cm. The larger errors occurred mainly when the fetal spine was not parallel to the film, and in the majority of the cases was on the negative side, the estimated length being less than the actual length.

An ossification center in the femoral epiphysis could be demonstrated in only two-thirds of the cases and in these it was a valuable aid. On the other hand, in some instances no ossification could be seen in the epiphysis although the fetus measured over 50 cm. This is in keeping with the observation that the fetus of a diabetic mother may, in spite of maturity, be retarded in its ossification.

Four roentgenograms; 3 graphs.

WILLIAM A. MARSHALL, M.D.
Chicago, Ill.

Integration of Cephalopelvimetry into an Obstetric Ward Service. Gerhart S. Schwarz. New England J. Med. **255:** 598–600, Sept. 27, 1956. (Columbia University College of Physicians and Surgeons, New York, N. Y.)

This report is a statistical analysis of the correlation of x-ray cephalopelvimetry to obstetric outcome in 350 patients measured by the Ball method. The x-ray diagnoses were classified according to the difference be-

tween the size of the fetal head and the maternal pelvis, as follows:

A. No radiologic disporportion.
B. Radiologic borderline disporportion.
C. High radiologic disporportion.

In 220 cases the radiologic classification was "A" and in 4 of these (less than 2 per cent) surgical intervention for disproportion was required. Thirty cases were diagnosed as "C," and in 25 of these (83 per cent) surgical delivery was necessary. Of 60 "B" cases 15 (25 per cent) required surgical delivery. Evidently, an x-ray diagnosis of "no disproportion" is very reliable; the accuracy of the "high disproportion" group is acceptable, but the group of borderline diagnoses would appear too large.

In 9 patients measurements were contrary to outcome (see Schwarz, Kirkpatrick, and Tovell: Radiology **67:** 854, 1956). The 60 cases of radiologic borderline disproportion must be counted as technical failures.

From the study it is concluded that cephalopelvimetry is useful because it proved capable of reducing the number of doubtful cases from 350 to 69 (approximately a fifth). Since roentgen cephalopelvimetry was used sparingly, *i.e.*, only in clinically doubtful cases, it was felt that the method had been given the severest test likely to be encountered in obstetric practice. Even with its deficiencies, the procedure was actually misleading in only 5 instances (1.4 per cent). This would not appear too serious a fault since the obstetrician does not rely on x-ray measurements alone.

Three "flow" charts. JOHN F. RIESSER, M.D.
Springfield, Ohio

X-Ray Pelvimetry by the Colcher-Sussman Method. A Study Comparing It with Stereoscopic Parallax Pelvimetry. D. W. Freeman. Minnesota Med. **39:** 583–585, September 1956. (St. Louis Park Medical Center, Minneapolis, Minn.)

A comparison was made of the results of x-ray pelvimetry with the Colcher-Sussman and the stereoscopic parallax (Hodges) methods. With the possible exception of the transverse diameter of the inlet, the variability in results obtained by the two technics was of the same order of magnitude as one might expect with repeated measurement of any pelvimetry roentgenogram by one person. The author recommends the simpler Colcher-Sussman method (Am. J. Obst. & Gynec. **57:** 510, 1949. Abst. in Radiology **54:** 303, 1950).

Four figures; 2 tables.

An X-Ray Pelvimetric Study of Relaxin Extract in Pelvic Expansion. Arthur Weinberg. Surg., Gynec. & Obst. **103:** 303–306, September 1956. (Far Rockaway, N. Y.)

It is common knowledge that, during pregnancy, changes take place at the pubic symphysis and to a lesser degree at the sacroiliac synchondroses which result in taking up of water by the cartilage and softening and separation of the joints, all of which lead to widening of the bony pelvic ring, facilitating delivery of the fetal head. Lately these changes have been ascribed to a third ovarian hormone called relaxin which is detectable only in pregnancy. It rises progressively following conception, reaches a plateau, and drops precipitously immediately following delivery.

The author investigated the role of relaxin in producing pelvic expansion. A total of 15 patients were studied, 4 non-pregnant and 11 pregnant.

The 4 non-pregnant females were selected because, in a previous recent pregnancy, they had manifested physiologic pubic symphysis separation. They were primed with estrogen for a two-week period, then given relaxin in saline intramuscularly over a three-day period. Pelvimetry was performed before the relaxin injections and twelve hours after the last injection. No symphysial separation or increase in the pelvic diameter was demonstrated.

The second series of 11 pregnant women were given the hormone in five daily injections of a repository form. Pelvimetry performed just before and just after delivery showed no significant changes from the patient's original measurements.

The author concludes that, under the conditions of his clinical trials, relaxin is not a significant factor in causing separation of the symphysis or pelvic expansion.

Two tables. FREDERICK J. MUNSON, M.D.
University of Pennsylvania

THE GENITOURINARY SYSTEM

Diatrizoate Sodium. New Urographic Contrast Medium. Everett E. Seedorf and Eldon O. Bradfield. J.A.M.A. **162:** 192–193, Sept. 15, 1956. (E. E. S., 405 Main St., Peoria, Ill.)

A comparison was made of diatrizoate sodium (Hypaque), recently introduced as a urographic medium, with sodium acetrizoate (Urokon). Two hundred patients received 30 c.c. and 50 patients, 20 c.c. of 50 per cent Hypaque; 100 patients, 20 to 25 c.c. and 50 patients 30 c.c. of 30 per cent Urokon; 50 patients 25 c.c. of 50 per cent Urokon. The authors concluded from their study that 30 c.c. of a 50 per cent solution of Hypaque produces urograms of superior quality in a greater percentage of patients than can be obtained with 20 to 25 c.c. of a 30 per cent solution of Urokon and slightly better urograms than were obtained with 25 c.c. of a 50 per cent solution of Urokon. This is thought to be attributable to the higher iodine content in the dosage of Hypaque. It is believed, also, that the slower elimination of this medium permits clearer visualization of the ureters.

There are few side-effects from Hypaque, particularly when the solution is not injected too rapidly. Objectionable reactions to this medium in doses of 25 c.c. of the 50 per cent concentration included vein-cramping, nausea, and urticaria.

Two tables. JOHN P. FOTOPOULOS, M.D.
Hartford, Conn.

Five-Year Results of Operation for Nephroma. Use of Diagnostic Exploration in Hæmaturia. A. Wilfrid Adams. Brit. J. Surg. **44:** 126–131, September 1956. (Bristol, England)

The author presents here the five-year results of 10 nephrectomies performed for neoplasms in adults, originally reported in 1950 (Brit. J. Surg. **38:** 210, 1950). Four patients are still alive without known recurrence.

The interest of the paper for the radiologist lies in the section headed "The Pyelographic Fallacy and the Prostatic Decoy." The author cites a case recorded in his earlier paper in which "disarmingly normal pyelography, both intravenous and retrograde" led to postponement of operation and a fatal termination. He reports here another example, in which a "lurking nephroma" was missed and hematuria was attributed to an adenomatous prostate. To quote from his summary:

"The disquieting delay in operating on nephroma results from a too implicit trust in data derived from routine urological investigation. *Negative findings are not infallible!* Despite them, the risk of tumours remains in a significant proportion. Diagnostic exposure of the kidney will settle the diagnosis and should be done early where grave suspicion of lurking nephroma remains. The risk attendant on surgical incision is in these days no greater than that incurred by expectant treatment."

Six roentgenograms: 6 photographs.

The Pelvic Ureter in the Male and Female. Gladys E. Wadsworth and Eduard Uhlenhuth. J. Urol. **76:** 244–255, September 1956. (Medical School of the University of Maryland, Baltimore, Md.)

The authors have demonstrated in 10 male and 10 female pelves that (1) the ischial spine is not a true landmark in the course of the pelvic ureter and (2) that the female pelvic ureter is approximately $^1/_2$ inch longer than the male. Difference in length of the female pelvic ureter is due to the position of the bladder, which lies more ventrally and caudally and closer to the inferior end of the symphysis pubis than in the male.

In the cadavers studied, the ureters were injected with barium paste and the vesicle orifices as well as the ureteral orifices were marked with lead pellets before lateral, craniocaudal, and ventrodorsal films were taken. Films revealed the ureteral angle approximately $1^1/_2$ inches cranial and ventral to the ischial spine. Measurements were then made on the films, using two standard lines at right angles to each other. The caudal border of the 5th lumbar vertebra was taken as the commencement of the pelvic ureter. After the exposure of the films the ureters were removed and measured directly.

Although this is a small series of cases the anatomical relationships demonstrated are significant.

Nine figures; 7 tables.

DECK E. CHANDLER, M.D.
University of Pennsylvania

Magnesium Dibasic Phosphate Identified as a Crystalline Component of a Urinary Calculus. Jonathan Parsons. J. Urol. **76:** 228–230, September 1956. (Edsel B. Ford Institute for Medical Research, Henry Ford Hospital, Detroit 2, Mich.)

The author reports the identification of a new magnesium phosphate compound ($MgHPO_4.3H_2O$) in the course of routine x-ray diffraction analysis of a renal calculus. The new compound occurred in combination with hydroxyl apatite.

Three diffraction powder patterns; 1 table.

Newer Roentgenographic Techniques in the Diagnosis of Retroperitoneal Tumors. John A. Evans and Nathan Poker. J.A.M.A. **161:** 1128–1132, July 21, 1956. (J. A. E., 525 E. Sixty-Eighth St., New York 21, N. Y.)

The authors emphasize the importance of specialized roentgen technics in the establishment of the diagnosis of retroperitoneal tumors. In a brief review of conventional roentgen methods, *i. e.*, plain films, gastrointestinal series, and pyelographic studies, they list established criteria: (1) deflection of the ureter, (2) distortion or compression of the renal collecting system, (3) renal rotation, (4) obscuration of the psoas shadows, (5) soft-tissue masses, (6) radiolucencies produced by lipomatous tumors.

Among the newer roentgenographic technics, translumbar aortography and nephrotomography afford means of identifying a renal mass with reasonable exactness. The authors believe that nephrotomography is safer than translumbar aortography. After establishing circulation time, they obtain an aortogram by rapid injection of a contrast medium (Urokon or Hypague) into the antecubital vein. Immediately following this, tomograms of the kidneys are taken.

With this procedure good accuracy in differentiating renal cysts from tumors has been achieved. The former appear as smoothly marginated, rounded lucent areas surrounded by opaque renal parenchyma. Tumors produce an area of similar or greater density than the adjacent normal parenchyma. If necrosis has produced cystic degeneration of a tumor, the translucency thus produced has ill-defined borders. An abnormal vascular bed is demonstrated in the preliminary film when tumors are present.

Presacral air injection, alone or in combination with nephrotomography, has proved useful in demonstrating extrarenal tumors. The use of pure oxygen is advised. The authors mention several possible pseudotumor defects which may lead to diagnostic error. The fundus of the stomach may be mistaken for a suprarenal tumor. The duodenum or an enlarged spleen may also cast a confusing shadow.

Six roentgenograms. ARTHUR SHUFRO, M.D.
University of Michigan

THE ADRENALS

Topical Diagnosis of Pheochromocytoma. H. Kindler and H. P. Kuemmerle. Fortschr. a. d. Geb. d. Röntgenstrahlen **85:** 330–333, September 1956. (In German) (Chirurgische Universitätsklinik Freiburg, Breisgau, Germany)

A pheochromocytoma is usually suggested by the occurrence of paroxysmal attacks of hypertension. Determination of the size of the tumor, however, and its exact localization may cause considerable difficulty, particularly since the tumor may lie outside of the adrenals or because more than one tumor may be present. According to the literature, these tumors involve one adrenal in 80 per cent of the cases, both adrenals in 9 per cent, the lumbar and paravertebral area in 6 per cent, and other locations (abdomen, chest, bladder, etc.) in 5 per cent.

In conformity with these statistics, efforts to locate the tumor should be concentrated mainly around the kidneys.

Of the various diagnostic procedures, the x-ray examination gives the most information. If the plain scout film is not informative, gastrointestinal roentgenography may show displacement of the intestinal organs, and the intravenous pyelogram may give important hints. The maximum of diagnostic information, however, is usually obtained by pneumograms of the retroperitoneal space following air insufflation by either the perirenal or the presacral route. Tomography is a routine procedure.

The authors report the case of a 16-year-old boy who had all the clinical signs of a pheochromocytoma. The tomographic films, after presacral air inflation, showed a tumor of the right adrenal and a second tumor in the

region of the pelvis of the left kidney. Both tumors were surgically removed, leading to a lasting cure.

Two roentgenograms.

WILLIAM A. MARSHALL, M.D.
Chicago, Ill.

MISCELLANEOUS

The Subphrenic Abscess: A Necessary Re-evaluation. Walter H. Gerwig, Jr., and Brian Blades. Ann. Surg. **144:** 356–364, September 1956. (George Washington University School of Medicine, Washington, D. C.)

In the era before the use of antibiotics, subphrenic abscess development was usually indicated by a relatively clear-cut clinical picture. Patients appeared critically ill, peritonitis would develop, and signs of concealed pus would become apparent. With the modern use of the antibiotics, the classical clinical findings have been camouflaged.

In a study of 9 patients with a history of abdominal surgery or perforated viscus with subsequent development of a subphrenic abscess there was an initial period of well-being or improvement. Ileus was usually present in the beginning but disappeared. Under antibiotic therapy, a relatively benign state may persist for several days, but escape from the influence of these drugs may be extremely rapid, demanding emergency surgical intervention.

One factor predisposing to the development of subphrenic abscess appears to be the tendency for free air to persist in the peritoneal cavity after upper abdominal surgery. In a side-study of 100 patients it became apparent that the slender individual was subject to a higher incidence and longer duration of pneumoperitoneum.

The development of an air bubble in the upper abdomen, demonstrable roentgenographically, is the most important diagnostic sign. When this tends to increase in size, intervention is absolutely indicated. Lack of fixation of the diaphragm and lack of significant pleural fluid are not felt to be assurance that subphrenic abscess is not present.

The anterior superior space and left subphrenic area seem to be affected more often than in the pre-antibiotic era. The most common pathogen in the authors' series was *Staphylococcus aureus*, which at present seems to be sensitive to Chloromycetin.

Seventeen roentgenograms; 1 table.

JOHN F. RIESSER, M.D.
Springfield, Ohio

Roentgenologic Investigation of Patients with Heterosexual Development. Theodore A. Tristan, Walter R. Eberlein, and John W. Hope. Am. J. Roentgenol. **76:** 562–568, September 1956. (Children's Hospital of Philadelphia, Philadelphia, Penna.)

The commonest type of heterosexual development is female pseudohermaphroditism secondary to congenital adrenal hyperplasia. Next in frequency is male pseudohermaphroditism of unknown cause. Much less common is female pseudohermaphroditism *not* secondary to congenital adrenal hyperplasia. True hermaphroditism is the rarest of all.

Since it is important to determine the predominant sexual development in order to establish how the child should be brought up, the roentgenologist can supply valuable information by outlining with an opaque substance the internal structures communicating with the external orifice. The authors describe their technic of studying the anatomy of the urogenital system. They use a blunt-end syringe, which is placed inside the urethral or urogenital orifice, cushioned against the perineum with pads of orthopedic cast sponge rubber. Iodochlorol or 30 per cent Urokon is the medium employed. The study is made with the patient in the lateral recumbent position, under fluoroscopic control. Urography may be necessary to confirm some of the anatomic findings. These are well described and illustrated.

Unfortunately, female pseudohermaphroditism, male pseudohermaphroditism, and true hermaphroditism may be associated with the same type of malformation of the external genitalia, and the findings on roentgen examination may be identical. The determination of urinary 17-ketosteroids will demonstrate an excessive excretion in cases of congenital adrenal hyperplasia; urinary excretion of pregnanetriol is also elevated in this condition. Chromosomal sex determination, obtained either by skin biopsy or buccal smears, may be useful.

Ten roentgenograms, 2 photographs; 1 table.

JOHN P. FOTOPOULOS, M.D.
Hartford, Conn.

TECHNIC

Experiences with Routine Utilization of New Roentgenographic Procedures in a Sanatorium. S. Wannovius. Röntgen-Blätter **9:** 265–274, September 1956. (In German) (Hamburgische Tuberkulose-Krankenhaus, Edmundsthal-Siemerswalde in Geesthacht, Germany)

The Usefulness of Paper Roentgenograms in Diagnostic Roentgenology. Herbert Schober. Röntgen-Blätter **9:** 274–280, September 1956. (In German (Hamburg, Germany)

The Employment of Hard Ray Technic in Paper Roentgenography of the Chest. O. Willbold. Fortschr. a. d. Geb. d. Röntgenstrahlen **85:** 510–515, October 1956. (In German) (Eckermannstr. 1, Bevensen, Lüneburger Heide, Germany)

Paper roentgenography has always been considered a second-best procedure when compared with the use of double-coated celluloid film, and this is still true. A recent trend has developed in Germany, however, leading to renewed interest in this method, especially for routine chest roentgenography. When high-kilovoltage technics are employed, it is said that the extrapulmonary shadows are less visible, the ribs appear more transparent, and cavities are more easily demonstrated. This should reduce the number of cases requiring body-section roentgenography.

The German Roentgen Society appointed a special committee, under the chairmanship of R. Prévôt, to study the problem. It concluded that the paper method is inferior to roentgenography with double-coated film, but is about equal to photofluorography, and certainly better than fluoroscopy. Paper roentgenography may be recommended in all cases where ultra-perfect contrast is not a strict necessity, and when the exposure may be repeated if necessary. The very stringent technical requirements, such as painstaking development, a high gloss (ferrotype), and adequate illumination for viewing, deserve special mention.

The basic reason for the use of paper roentgenography

is economy. In line with the recommendations of the above committee, paper may be considered acceptable in skeletal diagnosis for all orthopedic controls, but not for the first examination, especially when there is any question of tumor (metastatic or otherwise). With contrast media, especially barium, paper roentgenography is quite satisfactory, except when fine details must be brought out, as in certain cholecystographic and urographic procedures. In chest diagnosis, paper is adequate for estimation of heart size and shape, and for routine rechecks of both pathologic and non-pathologic conditions. Film is needed to confirm the presence or absence of small foci (silicotic, tuberculous, etc.) For chest examinations in children, celluloid should be used exclusively, to avoid any necessity of repetition of exposure. Some surveys may be performed with paper, but for persons known to have had contact with tuberculosis, the details available only on double-coated films are necessary for the detection of early minimal changes.

Paper has also been employed for both serial and simultaneous tomographic technics, although it is doubtful whether enough contrast was obtained for proper evaluation.

E. R. N. GRIGG, M.D.
Cook County Hospital, Chicago

A Photographic Device Attached to the X-Ray Apparatus (Combined X-Ray and Photographic Procedure). R. Bimler. Fortschr. a. d. Geb. d. Röntgenstrahlen **85:** 338–344, September 1956. (In German) (Hermann-Schramm-Unfallkrankenhaus, Murnau/Obb., Germany)

X-ray apparatus which is used mainly for orthopedic work or for industrial injuries, should be provided with an attachment for simultaneous photography, thus saving the expense of a special photographic department and obviating the necessity of a second examination with its attendant inconvenience and discomfort.

The author has been using a special gadget consisting of a miniature camera (Leica, Contex, or, preferably, a Robot) and two focusing lights (either photoflash or strobe) attached to the arm that carries the x-ray tube. All x-ray films are taken at a focal spot-film distance of 100 cm. and the lens of the camera, which is in the same horizontal plane as the anode of the x-ray tube, is focused at the same distance. The camera thus covers the full 14 × 17-inch film. The illuminating lights are strong enough to permit cutting down the lens diaphragm to f:11. In actual operation, a lead marker is first placed over two opposite corners of the cassette or film holder. The injured limb is properly positioned and the x-ray exposure is made. Then, without moving the patient, the tube is pushed sideways so that the camera occupies the place of the tube and the photograph is taken.

The author presents striking combinations of roentgenograms and photographs of injured hands before and after surgery, showing the defects of the soft tissues as well as of the bony structures in their proper relationship on a single picture. This is accomplished as follows: The miniature photographic film is placed in the photographic enlarger and the x-ray film is placed on an easel below this. The enlarger is focused in such a manner that the lead markers of the photographic film are exactly projected upon the corresponding markers on the x-ray film. Then, without moving the easel, a photographic paper the size of the x-ray film is placed under it, and the ordinary ceiling light of the dark room is turned on long enough to produce a light contact print of the skeleton through the x-ray film. This is then removed without disturbing the position of the photographic paper and the image of the miniature film is printed by means of the enlarger on the same paper. If the lead markers are properly superimposed, the two exposures will be in perfect register and the picture will show a true relationship between the bones and the scars of the skin and other soft-tissue changes.

Five photographs; 2 roentgenograms.

WILLIAM A. MARSHALL, M. D.
Chicago, Ill.

Mobile Radiotranslucent Litter-Table with Bucky Diaphragm. Joseph Stein, Ruth Hammerschlag, and Maxwell H. Poppel. J.A.M.A. **162:** 193–194, Sept. 15, 1956. (J. S., 130 W. Kingsbridge Road, Bronx 68, N. Y.)

The construction of a mobile radiotranslucent litter-table with a Bucky diaphragm is described. The table has a radiotranslucent top mounted on solid legs at the extreme ends and is equipped with lock wheels. A radiotranslucent foam rubber pad is provided for the patient's comfort. An extension at one end of the table allows for placement of a cassette for lateral skull projections without turning the patient. The metal trim around the table top permits the use of magnetic cassette holders. Two vertical rods are located below the table top, one at each end; running between these is a horizontal unit with two rods supporting the Bucky diaphragm, which can be moved on these rods and travel the table length. This horizontal assembly can be raised and lowered for magnification studies.

The use of the table has eliminated shifting of patients from bed to stretcher to x-ray table and back again. The table may be used in the X-ray Department as an accessory for special positioning.

Two photographs. JOHN P. FOTOPOULOS, M.D.
Hartford, Conn.

Tetramethylene-hematoxyline for the Elimination of Colonic Gas in Roentgen Examination of the Abdomen. Mario Di Egidio. Arch. di radiol. **4:** 539–547, 1955. (In Italian) (Ospedale Reale di Taiz, Yemen)

The author is a radiologist in a hospital located in Taiz (Yemen). His patients are of Arabian extraction and suffer frequently from nephrolithiasis, which makes pyelography one of the most common examinations. These patients very often have large amounts of gas in the colon, partly because of the high incidence of parasitic disease of the intestine but mainly because the population is addicted to the leaves of *Catha edulis*, a tropical plant which contains an alkaloid (*cathine*), said to provoke a styptic atony of the gastrointestinal tract.

After trying various preparations to overcome this difficulty, the most encouraging appeared to be tetramethylene-hematoxyline (marketed by Lepetit under the name of *Almateine*). Eight tablets dissolved in 3 liters of water are given as an enema just prior to the examination. In patients with severe constipation, 20 gm. of castor oil may be given one day prior to the actual examination, except for cholecystography, in which the enema is employed exclusively. When in weak alkaline solution, tetramethylene-hematoxyline splits into formaldehyde (a disinfectant) and hematoxyline (an astringent).

The method was used in about 400 patients, and it is said to result in significant reduction of colonic gas.

Seven roentgenograms. E. R. N. GRIGG, M.D.
Cook County Hospital, Chicago

On the Thickness of the Layer in Rectilinear Tomography. Olof Willner. Acta radiol. **46:** 511–517, September 1956. (University Hospital, Upsala, Sweden)

The author believes that the thickness of the layer in rectilinear tomography should be calculated as a mean value of half the amplitude, taking into consideration not only the permissible tomographic unsharpness, but also the more or less poor contrast produced by the blurred detail of the object.

This paper contains diagrams and various mathematical formulas supporting this method in determining the thickness of the layer in focus during tomography and does not lend itself well to being abstracted.

The calculation of the thickness of the layer by rectilinear tomography is not only of theoretical interest but also is of practical value in determining the intervals between the tomographic sections of the object to be examined and for localizing foreign bodies by tomography. J. P. CHAMPION, M.D.
Grand Rapids, Mich.

RADIOTHERAPY

Carcinoma of the Lip. Paul M. Burke and Frederick S. Hopkins. New England J. Med. **255:** 552–555, Sept. 20, 1956. (Westfield State Sanatorium, Westfield, Mass.)

The authors report on 182 patients treated primarily for carcinoma of the lip. One hundred seventy-six of these patients were males. Eighty-six of the tumors were less than 1.5 cm. in diameter; 85 were between 1.5 and 4 cm., and 11 were recorded as being larger than 4 cm.. Sixty-nine patients had surgical excision of their tumors; 97 were treated solely by irradiation; in 16 surgical and x-ray therapy were combined. The larger the lesion, the greater was the tendency to prefer irradiation over surgical excision.

For the neoplasms of less than 1.5 cm. diameter, the five-year survival results for surgery and irradiation were almost identical, 74 and 75 per cent (uncorrected), respectively. For tumors between 1.5 and 4.0 cm., the corresponding figures were 54 and 56 per cent. Only 1 of the larger tumors was treated surgically and the patient remained alive after six years. Eight of the remaining patients in this group were treated by irradiation, with 3 five-year survivals. The tendency was to treat the lesions of higher grade malignancy with radiation rather than surgery.

Twelve patients had metastases in the lymph nodes of the neck at the original examination. In 19 of the remaining 170 patients, metastatic disease later developed in the cervical nodes. In 3 of the 19 the primary lesion was excised surgically; 13 had received x-ray therapy, and 3 had received both irradiation and surgical therapy.

In 16 cases pathologically proved metastatic carcinoma was found in the surgical specimen after neck dissection. Seven survived five years and were apparently free of disease at the time of the report, a clinical cure rate of 44 per cent. In 7 patients no tumor was found in the resected specimen by the pathologist after neck dissection was performed with a clinical diagnosis of metastatic carcinoma. All 7 patients survived for five years without recurrence.

Eighteen patients received x-ray therapy to clinically positive lymph nodes in the neck. Of this number, 2 had survived for five years without recurrence. One of these patients underwent upper neck dissection two years after the radiation therapy to the neck, and the surgical specimen revealed metastatic disease.

The authors discuss the opinions of various groups as to the best procedure for the primary lesion of the lip. Their own results suggest that cancers of similar histologic grade and size do approximately as well after surgical as after x-ray therapy. They also believe that routine prophylactic neck dissection should not be done, since the occurrence of lymph node metastases is only about 10 per cent after treatment of the primary lesion and even some of these patients may be saved by therapeutic neck surgery.

Four tables. JOHN P. FOTOPOULOS, M.D.
Hartford, Conn.

Carcinoma of the Nasopharynx. Treatment with Radioactive Cobalt. Wayne W. Deatsch. California Med. **85:** 180–182, September 1956. (2075 Hayes St., San Francisco 17, Calif.)

Report is made of 22 patients receiving radiocobalt therapy for carcinoma of the nasopharynx from 1949 to 1954. Three-quarters of these patients had metastatic disease at the time of first examination. The most frequent initial symptoms were enlarged cervical nodes and nasal obstruction or a bloody nasal discharge. In 10 per cent of the cases, there was evidence of intracranial extension of the disease, with cranial nerve involvement at the time of the original examination. The disease was three times as common in males as in females and occurred most frequently in the middle age groups, 71 per cent of the patients being between thirty and fifty-nine.

The first step in treatment is a posterior septectomy from the level of the middle turbinate to the floor of the nose, performed with the patient under general endotracheal anesthesia. At the same time, visible portions of the tumor in the nasopharynx are cauterized. A bead of radioactive cobalt contained in a Foley bag catheter is introduced into the nasopharynx through the nose. The bag of the catheter is then inflated and the position of the radiating source adjusted by traction on the catheter and/or nasal packing. A surface dose of 2,000 to 6,000 gamma roentgens is given. All patients then receive "full courses of external x-ray therapy to the nasopharynx" and also to cervical fields if palpable nodes are present. Radioactive cobalt therapy is considered supplemental to adequate external roentgen irradiation.

All 3 patients in the series with localized lesions were alive at the time of this report, 2 for more than five years. Five of 11 patients with metastatic disease were alive after varying periods. Of 8 patients treated for recurrent disease following irradiation, 2 survived.

Two tables. JAMES W. BARBER, M.D.
Cheyenne, Wyo.

Experimental Treatment of Recurrent Carcinoma of the Nasopharynx with Electrodesiccation, Radioactive Cobalt and X-Ray Radiation. Francis A. Sooy. Ann. Otol., Rhin. & Laryng. **65**: 723–735, September 1956. (490 Post St., San Francisco, Calif.)

In an effort to afford relief to 6 patients with carcinoma of the nasopharynx recurrent following extensive external roentgen irradiation, an additional course of treatment was given, combining intranasal septal resection, electrodesiccation, and radioactive cobalt. Following septectomy and electrodesiccation of the tumor and adjacent mucosa of the nasopharynx, a radioactive cobalt bead in a Foley type bag catheter was introduced through the nose. The bag was filled with Diodrast and held in position with anterior and posterior nasopharyngeal packs. Filming aids calculation of dosage and allows verification of proper placement of the radiating source. Each patient was given 4,000 to 6,000 gamma roentgens in two divided applications one week apart. Dosage was calculated at a surface 1 cm. outside the catheter bag.

Three of the 6 patients with advanced recurrent disease remained tumor-free at the time of the report for periods of more than six years. The other 3 died within a year following the additional therapy, but in 1 of these no tumor was found in the region of treatment. Patients with involvement of the base of the skull by the tumor do not respond to this treatment.

Postoperatively and immediately following radiotherapy there was considerable crusting and radiation mucositis in and around the treatment zone, requiring six to eight weeks for clearance. In most instances small local areas of bone sequestration occurred, particularly in the anterior sphenoid and posterior ethmoid areas. Severe atrophic rhinitis or ozena, a feared complication, failed to develop in any of these patients. Dry adhesive otitis media occurred in 2 cases, with impairment of hearing in 1. Local tumor recurrence is sometimes difficult to differentiate from chronic granulation resulting from the treatment. The author has found nasopharyngeal swabs with studies of exfoliated cells very helpful in these instances.

Since the initial study an additional 10 patients have been managed by this method with a 50 per cent survival rate over periods ranging from six months to four years. It is stressed that in all cases the primary treatment must be external roentgen irradiation.

Three anatomical drawings illustrate the technic.

JAMES W. BARBER, M.D.
Cheyenne, Wyo.

The Treatment of Malignant Melanoma with Special Reference to the Possible Effect of Radiotherapy. Lorentz Nitter. Acta radiol **46**: 547–562, September 1956. (Norwegian Radium Hospital, Oslo, Norway)

Wide differences of opinion exist as to the proper therapy for malignant melanoma, some holding that radical surgical excision is the only proper form of treatment, while others claim good results for radiation therapy. A third view favors a combination of surgery and radiotherapy.

The author reviews a series of 192 verified cases seen at the Norwegian Radium Hospital in 1932–48. The majority were treated by rather conservative surgery followed by irradiation.

In Stage I cases (no lymph node involvement clinically) results with limited surgery and postoperative irradiation were approximately the same as those reported by Pack, who relied on radical surgical excision. Thirty-five of 107 patients were alive after five years. Prophylactic treatment of the regional nodes is recommended for lesions in this stage, as a fairly high percentage of these patients have node involvement, even though it is not clinically detectable. The choice between surgical dissection and irradiation for this purpose depends upon the patient's age, general condition, and the location of the primary tumor in relation to the nodes.

There were 42 Stage II cases (regional node metastases limited to one group of nodes). Of the 12 survivors in this group, 10 received irradiation of the node metastases as the principal form of therapy. In 9 of the 12 patients metastasis was verified by biopsy; in the remainder only clinical evidence was available.

In Stage III (with more widespread disease) none of the 18 patients lived five years.

The conclusion is reached that limited surgical excision of the primary tumor followed by radiotherapy of the scar region and of the lymph node area produces as good results as radical surgery.

Two photographs; 9 tables. J. A. GUNN, M.D.
Grand Rapids, Mich.

Cancer of the Cervix, An Appraisal. Joe Vincent Meigs. Am. J. Obst. & Gynec. **72**: 467–478, September 1956. (226 Marboro St., Boston, Mass.)

The author believes that irradiation and surgery both have a place in the treatment of cancer of the cervix but that one or the other should be the primary procedure. The combination of full radiotherapy (radium and x-rays) and radical operation he regards as a serious mistake. His present appraisal is based upon a series of 131 cases (Stages I and II) operated upon between 1940 and 1949, with a five-year cure rate of 73 per cent. For comparison he presents the figures from the Vincent Memorial Hospital and the Pondville Hospital (Massachusetts Department of Public Health), as published in the *Annual Report*, showing a 55.0 per cent five-year survival for cases of Stage I treated with radiation alone and 74.0 per cent for those treated by surgery. In Stage II the figures were 44.0 per cent with radiation and 51.0 per cent with surgery. From this it appears that in his own series surgery helped the total result though it is to be borne in mind that the patients operated upon were probably the better risks.

Evaluation of the two methods of treatment is difficult, especially when they are used in combination. Some conclusions are reached, as follows: The surgical results in the group are good. Radiation did not help statistically but did cure 3 of 10 surgical failures. Patients who had planned preoperative irradiation had the smallest percentage (8 out of 49) of positive nodes; 4 of these have survived five years. The cure of patients with positive nodes is excellent, running from 31 to 55 per cent in the five-year group to 25 to 40 per cent in the ten-year group. On the whole the patients with radiation seem to do about the same as the others in this group. Fistula formation is too frequent, averaging from 8.4 to 15.0 per cent. Pelvic irradiation apparently had little effect on its frequency in this series.

It is conceivable that surgery will cure irradiation failures in a fair percentage of cases and that irradiation will cure some of the surgical failures. This indicates the importance of selection.

Nine tables. ROBERT L. EGAN, M.D.
University of Texas, Houston

Urinary Tract Fistulas Following Radical Surgical Treatment of Carcinoma of the Cervix (Exclusive of Exenterations). Alexander Brunschwig and Henry Clay Frick, II. Am. J. Obst. & Gynec. **72:** 479–488, September 1956. (Memorial Center for Cancer and Allied Diseases, New York, N. Y.)

From September 1947 to Dec. 31, 1950, 212 operations envisioning cure for carcinoma of the cervix of Stages I, II, III, and IV were performed at the New York Memorial Center. This number includes patients with previous surgery or irradiation and 19 instances of carcinoma of the cervical stump. The selection of patients was minimal. The surgical procedure envisaged was always a radical panhysterectomy with pelvic node dissection, but if that was regarded as too severe for the patient, then a Wertheim or Schauta operation was done.

In 50 patients urinary tract fistulas developed postoperatively. In only 18 of this number was the primary disease limited to the cervix, and in 4 of these it had been completely eradicated by irradiation. Nineteen patients had invasion of the vaginal fornices or parametria; in 6 the bladder and/or ureters were involved, and in 7 there were peripheral pelvic node metastases.

There were 27 ureteral fistulas and 23 vesical fistulas. Of 28 patients who did not survive, 25 died of cancer in less than five years. Of 22 surviving, 11 are urological cripples with nephrostomies, cutaneous ureterostomies, persistent fistulas, or severe incontinence without "ostomies." Two deaths were attributable to reparative surgery.

Contrary to the widely held view that previous irradiation tends to increase the likelihood of urinary fistulas if operation is carried out, this series indicates that irradiation received prior to surgery did not contribute to fistulas on the basis of ischemia.

While lower urinary tract fistulas are a complication to be expected in radical surgical management of cervical cancer, they do not pose a serious deterrent to such treatment.

Eight tables. ROBERT L. EGAN, M.D.
University of Texas, Houston

Betatron Therapy in Advanced Carcinoma of the Urinary Bladder. Justin J. Cordonnier and William B. Seaman. J. Urol. **76:** 256–262, September 1956. (Washington University School of Medicine, St. Louis, Mo.)

Therapy with the betatron allows a greater dose to be administered to deep lesions because there is better limitation of the beam due to less scatter and because of the minimum skin reactions produced with relatively large depth doses. It was estimated from reports in the literature that the relative biologic effect of betatron therapy was 0.6 to 0.8 compared to 200-kv therapy. In order to compensate for this the authors used larger doses but now feel that their experience has not borne out this relationship.

Seventeen patients with advanced carcinoma of the urinary bladder in which cure by radical surgery or conservative radiation therapy was deemed impossible were treated with 4,700 to 7,500 r over a period of twenty-three to thirty-nine days through three portals. In 4 patients there was apparent cure of the local disease, but 2 died of either metastases or complications. The remaining 2 patients were alive without evident disease after fourteen months. Seven patients had good palliation of bladder symptoms, but several of these had other complications of therapy. Aggravation of symptoms occurred in several cases, and in 2 patients operations to divert the urinary stream became necessary.

All patients had cramps and diarrhea starting in the third week of therapy and continuing for two weeks after its completion. Nine patients experienced major gastrointestinal complications, *i.e.*, intestinal obstruction, intestinal perforation, intractable diarrhea or fistula formation. These complications would have been avoided with lower doses, but the authors intentionally used radical therapy to see whether any salvage of these advanced cases was possible. They feel that their experience, along with the experience of others reported in the literature, shows that there is still no cure for advanced carcinoma of the bladder and indicates that treatment in these cases should be limited to palliation. Curative therapy with the betatron still has a place in early bladder carcinoma when the patient refuses radical surgery.

One table. LESLIE M. ZATZ, M.D.
University of Pennsylvania

Automatic Control of the Tube Current as a Means of Dose Regulation in Tangential Rotation. H. Holthusen, F. Gauwerky, and F. Heinzel. Brit. J. Radiol. **29:** 274–276, May 1956. (St. George's General Hospital, Hamburg, Germany)

In tangential rotation therapy uneven dosage occurs over surfaces such as the thorax because, in the region where the angle of the beam is at or near 90°, the tissues are exposed to direct irradiation for a shorter time than the fully irradiated areas. Also the skin near the sternum (in the case of thoracic irradiation) is exposed longer to direct irradiation as a leads to an attempt to reach the retrosternal space. This leads to a sharp decline in dose extending from the sternal region to the axilla.

To compensate for this, either the speed of the x-ray tube movement or the rate of dose output can be varied. The latter effect can be achieved by regulating tube current in accordance with a predetermined scheme, with less constructional difficulties, and is therefore the method of choice.

The tube current is reduced in the higher dosed skin areas and increased in positions of the tube in which the surface areas receive lower doses. To accomplish this, a regulating template is interposed between a light slot and a photo-electric cell. The disk rotates over the light slot synchronously with the pendulum movement of the x-ray tube. The dose rate can thus be varied as desired by varying the tube current.

Four figures. RICHARD P. STORRS, M.D.
Los Angeles, Calif.

Radiation Dosimetry Using Alkali Halide Crystals and the Absorption of Betatron Bremsstrahlung in Water and Bone. D. Kahn. Acta radiol. **46:** 563–569, September 1956. (Norwegian Radium Hospital, Oslo, Norway)

Dosimeters for electromagnetic radiation are designed either to record instantaneous dose rate or to integrate the dose rate over the time of exposure. The simultaneous indicating instrument is prone to suffer from the effect of the intense radiation field on parts of the dosimeter other than the detecting element, such as insulators, signal leads, etc. The integrating type of

dosimeter either requires extensive manipulation, as is the case with the chemical type, or has the disadvantage of a somewhat limited range, as is true of a thimble chamber.

The use and behavior of alkali halide crystals in measuring roentgen and gamma radiation were investigated. Alkali halide crystals, when irradiated with roentgen and gamma rays, exhibit a wide variety of phenomena which may be used to indicate and measure radiation. Under irradiation, an electron can be ejected from a halide valence band and trapped at a negative ion vacancy, forming an F-center. If the crystal is exposed to visible light while in an electric field, the trapped electrons are liberated and produce a net drift before being permanently trapped at neutral halogen atoms or returned to the valence band in some other way. Measurement of this current, and hence the F-center density, gives a measure of the radiation dosage received by the crystal.

Dosimeters containing single crystals and powdered crystals of potassium bromide (KBr) were constructed and irradiated with 175-kev roentgen rays and 31-MEV gamma rays "bremsstrahlung" from the betatron at the Norwegian Radium Hospital. The response of these dosimeters relative to the Victoreen type thimble chamber was found to be approximately five times greater when irradiated with 175-kev roentgen rays than when irradiated by 31-MEV "bremsstrahlung". The response ratio was unchanged by lowering the maximum energy of the betatron from 31 MEV to 16 MEV.

Measurements of the linear absorption coefficient of 31-MEV "bremsstrahlung" in water and bone were carried out. The results agreed with the values obtained with an air chamber in the case of water and were in agreement with theoretical expectations in the case of bone.

Four drawings.

J. P. CHAMPION, M.D.
Grand Rapids, Mich.

Scintillation Counter for Beta-Ray Dosimetry. Paul N. Goodwin. Nucleonics **14:** 120–123, September 1956. (U. S. Public Health Service Hospital, Baltimore, Md.)

A scintillation counter is described capable of measuring isodose distributions from beta-emitting sources. A plastic scintillation material ($2 \times 2 \times 0.5$ mm.) is used to determine the relative energy absorption at various depths from plane sources.

Depth doses were measured by introducing polystyrene or Lucite spacers between source and detector. To measure the lateral distribution of dose the scintillator can be moved in a slot in a Lucite disk 0.5 mm. in thickness. The photomultiplier current was measured directly with a sensitive galvanometer.

Depth doses for three beta-emitting isotopes are given. These follow an equation of the form $(1/r^2)e^{\mu r}$ at depths greater than about 2 mm. Isodose curves are given for a plane circular Sr^{90} - Y^{90} source.

Five figures.

ROSS GARRETT
Memorial Center, New York

RADIOISOTOPES

Radiography with Thulium 170 and Xenon 133. W. V. Mayneord and H. J. D. Ireland. Brit. J. Radiol. **29:** 277–285, May 1956. (Institute of Cancer Research, Royal Cancer Hospital, London, S. W. 3, England)

Radioactive isotopes emitting low-energy gamma rays and x-rays may have a place in human radiography and in certain industrial applications. The authors have investigated the possibilities of radioactive thulium and xenon.

Mayneord previously described the physical problems involved in the use of thulium (Tm^{170}), whose primary radiation consists of high-energy beta rays and low-energy gamma and roentgen rays, and reported preliminary experiments on its application to radiography (Lancet **1:** 276, 1952. Abst. in Radiology **60:** 152, 1953. Also Brit. J. Radiol. **25:** 517, 1952) The source used in present work was a standard cylinder of sintered thulium oxide weighing 35 mg., activated in a heavy water pile by neutron flux. The active pellets were sealed in a double light alloy capsule to prevent leakage of the dangerous powder and just sufficiently thick to stop the primary beta rays. With this source a satisfactory radiograph of a human hand was obtained at a film-source distance of 10 cm. in an exposure time of forty-five seconds. Stronger sources could reduce the time required, but with such large sources the penetrating bremsstrahlung results in loss of contrast. The results obtained by this method do not approach the quality of a normal roentgenogram but are still sufficiently good for fracture examination under field conditions when no electrical supply is available.

Xenon 133 has a half-life of 5.27 days, a disadvantage which could be overcome by arrangement for a continuous supply of sources. In the event of accidental contamination the short half-life would actually be an advantage. In addition, xenon is an inert gas not easily absorbed in the body, while the radioactive isotope breaks down to a stable form of cesium.

The xenon 133 used in this study was obtained by solution of a freshly irradiated uranium slug. The separated xenon was adsorbed onto activated charcoal at the temperature of liquid nitrogen. Several curies of xenon 133 prepared in this way can be contained in an aluminum tube 3 mm. in diameter at room temperature. With such a source, a satisfactory radiograph of a hand can be made with a three-second exposure at 22 cm. distance six days after sealing of the source.

These radiographic sources are likely to prove clinically valuable, especially if combined with polaroid developing, for field work in time of war and for accidents where portability and freedom from supply mains are major factors and where fine detail is less important than detection of gross abnormalities, such as fractures, dislocations, and opaque foreign bodies in the tissue.

In practice two types of source would be needed, one of high radioactive content for large focal distances and a second of lower content and very small size for use at short focal distances, as in dental radiography.

In circumstances in which its short half-life is not a serious disadvantage, xenon 133 appears to give more satisfactory radiographs than does thulium 170 and is less hazardous in use.

Nineteen radiographs; 3 photographs; 3 diagrams.

RICHARD P. STORRS M.D.
Los Angeles, Calif.

I^{131}-Labeled Fat in the Study of Intestinal Absorption. Julian M. Ruffin, William W. Shingleton, George J. Baylin, Jacqueline C. Hymans, Joseph K. Isley, Aaron P. Sanders, and M. Frank Sohmer, Jr. New England J. Med. **255:** 594–597, Sept. 27, 1956. (Duke University School of Medicine, Durham, N. C.)

Curves of radioiodine blood levels have been established for normal persons after ingestion of an I^{131}-labeled test meal, whether radioactive-tagged albumin or radioactive-tagged fat. Less than 2 per cent of radioactive material is recovered in the total feces passed within forty-eight hours after ingestion of a fat test meal. This method of study is here applied to groups of patients presenting functional disorders, a variety of gastrointestinal diseases, and those who have had previous surgery for peptic ulcer. The fat test meal was employed.

A "cold" emulsion and a "hot" emulsion were used for this study. The "cold" emulsion consisted of 200 ml. of peanut oil, 200 ml. of water, and 15 ml. of Tween 80; the "hot" emulsion of 45 ml. of peanut oil, 5 ml. of I^{131}-labeled glycerol trioleate (2.0 mc of I^{131}), 50 ml. of water, and 4 ml. of Tween 80.

Each patient in a fasting state was given a test meal prepared as follows: To that volume of "hot" emulsion containing 25 microcuries of I^{131}, was added sufficient "cold" emulsion to make a total of 1 ml. per kilogram of body weight. Twenty grams of barium sulfate was added, and the entire contents mixed. The container was rinsed twice with tepid water and this also was ingested. The container was assayed for residual radioactivity. Twenty drops of Lugol's solution were given.

Blood samples were drawn at hourly intervals after the test meal and each sample assayed in a scintillation crystal-well counter. Total blood volume was assumed to be 7.2 per cent of body weight. With use of measured radioactivity of 2-ml. blood samples and calculated total blood volume, the total blood level of radioiodine was determined and expressed as percentage of the ingested material. Curves were constructed giving the relation of instantaneous I^{131} blood levels to time.

All stools passed in the succeeding forty-eight hours were collected in individual containers and assayed for radioiodine content with the scintillation crystal detector. The amount of radioiodine recovered in feces was expressed in terms of per cent of ingested material.

One hundred eight patients were studied and the determinations were subjected to statistical analysis. In 44 patients with apparent functional disorders, blood levels and fecal content were normal. Patients with small-bowel disease or pancreatic disease, numbering 41, invariably showed impairment of fat absorption. In cases of sprue in clinical remission blood and fecal values were normal. Diseases of the colon (16 patients) did not interfere with fat absorption. Of 79 patients who had undergone surgery for peptic ulcer, 53 per cent had significant impairment of fat absorption. This last group is reported separately (see following abstract).

This method of study offers a simple, accurate, and reliable test of fat absorption from the gastrointestinal tract. Abnormal values in the absence of previous gastric surgery, small bowel disease or pyloric obstruction, are suggestive of pancreatic disease, either pancreatitis or carcinoma.

Seven graphs; 1 table. JOHN F. RIESSER, M.D.
Springfield, Ohio

A Study of Fat Absorption after Gastric Surgery Using I^{131} Labeled Fat. William W. Shingleton, George J. Baylin, Joseph K. Isley, Aaron P. Sanders, and Julian M. Ruffin. Ann. Surg. **144:** 433–440, September 1956. (Duke University School of Medicine, Durham, N. C.)

An appreciable number of patients lose weight and become malnourished following the surgical procedures currently employed for peptic ulcer. The present report is intended to cast some light on fat absorption in these patients with correlation of type of operative procedure and change in weight. The method of study is describedin the preceding abstract.

A group of 84 patients were subjected to the procedure one to six years after partial gastrectomy (Billroth I, Hofmeister, and Polya types) or vagotomy with gastroenterostomy. The incidence of abnormal blood levels and fecal excretion of radioactive I^{131}-labeled fat was somewhat less in the Billroth I group than in those treated by the other procedures. A majority of patients who were under ideal weight were found to excrete higher amounts of fat in the feces than normal. A smaller percentage of patients were under ideal weight following vagotomy with gastroenterostomy than with other procedures. Impairment of fat absorption, which occurs in many patients following operative procedures on the stomach, seems to be an important factor in inability to regain preoperative weight and strength.

The validity of the radioactive labeled fat technic for study of fat absorption appears well established. Chemical analysis has shown the radioactive iodine to be attached to the fat fraction of blood and feces after administration of the fat test meal.

Two graphs; 2 tables. JOHN F. RIESSER, M.D.
Springfield, Ohio

Transfer of Radiosodium to Human Foetus in Late Pregnancy. C. G. Clayton, F. T. Farmer, and Trevor Johnson. Lancet **2:** 539–544, Sept. 15, 1956. (C. G. C., Royal Victoria Infirmary, Newcastle upon Tyne, England)

In normal pregnancy placental efficiency reaches its peak by the thirty-sixth week of gestation and diminishes progressively thereafter. This has been held responsible for the increased fetal mortality when pregnancy is prolonged for more than fourteen days after the expected date of confinement. The death of the fetus is attributed to intra-uterine anoxia. The best time for intervention to save these anoxic infants is difficult to decide from ordinary clinical signs, and a practical method of assessing placental function before the onset of labor will introduce new precision into the management of such cases.

If a tracer dose of radiosodium (20 μc of Na^{24} in 5.0 ml. of isotonic saline solution) is injected intravenously in the later weeks of pregnancy, the rate of accumulation of the isotope within the fetus can be observed with a collimated scintillation counter placed over the center of the anterior aspect of the uterus. The measurement of placental efficiency by a counter thus placed allows an almost unlimited number of observations to be made on the fetus instead of a single measurement at the time of delivery. The procedure can be employed repeatedly antepartum in any individual case and with complete safety and relatively minor discomfort to the mother. The total exposure to radiation of mother and fetus after the dosage of radiosodium used by the authors is said to be about 0.01 rad.

This method yields valuable information in cases of intra-uterine death from insidious injury to the placenta in the later weeks of pregnancy. It seems unlikely, however, that the abrupt deterioration of placental action which accompanies accidental hemorrhage will be as readily predicted, unless there is preceding pre-eclampsia. The method has proved reliable for diagnosing fetal death before this is possible radiologically.

Further developments in the useful application of radioactive substances to the problem of placental physiology will depend on close collaboration between obstetrician and physicist.

Nine figures.

Apparatus for Injecting Radioactive Colloids. J. P. Vigne, R. Santelli, and J. Fondarai. Nucleonics **14:** 110, September 1956. (Laboratoire des Isotopes, Centre de Lutte contre le Cancer, Marseilles, France)

Injection of colloidal radiogold presents several problems: (1) preparation of a suitable dilution; (2) injection of an accurately measured volume; (3) use of the same apparatus on several patients so as to reduce the number of manipulations; (4) easy decontamination of the apparatus.

To meet these requirements, the authors have used a modification of a special blood transfusion apparatus. This consists essentially of a pump which provides continual movement of the liquid by pinching a turn of rubber pipe against a cylinder with a rotating wheel. The volume of liquid passed through the pump is measured by the number of wheel revolutions.

The radiogold solution is made up by first pumping the gold colloid into a shielded bottle. Then enough physiological serum is added to obtain the desired dilution. For use, the pump flow is reversed and the output connected to the injection trocar.

The apparatus has several advantages: it can be remotely controlled by the motor drive; the rubber tube, being very thin, makes radiation from unshielded parts low, and decontamination is easy.

One figure. ROSS GARRETT
Memorial Center, New York

The Comparative Effectiveness of Four Beta-Emitting Isotopes Fed to Habrobracon Females on Production and Hatchability of Eggs. Daniel S. Grosch, Robert L. Sullivan, and Leo E. LaChance. Radiation Res. **5:** 281–289, September 1956. (D. S. G., Department of Genetics, North Carolina State College, Raleigh, N. C.)

Permanent sterility of female wasps was obtained after feeding P^{32} or Sr^{89} but not after feeding S^{35} or Ca^{45}. Furthermore, as shown by lowered egg production and hatchability, the descending order of effectiveness of the four ingested radioisotopes was the same as the ascending order of their physical half-lives. Except with Ca^{45}, results reflect a greater influence on the number of eggs produced than on the viability of embryos.

Technics included feeding isotopes in honey mixtures and weighing the female wasps on a precision balance before and after feeding. Biological features, such as egg incorporation of the isotope, as well as physical features, such as energy and intensity of the emissions, are considered in the discussion.

Four graphs; 1 table. AUTHORS' ABSTRACT

RADIATION EFFECTS

The Hazards from the Increasing Use of Ionizing Radiations: A Symposium. I. The Control of Radiological Hazards in the Future Development of Atomic Energy. W. G. Marley. Brit. J. Radiol. **29:** 261–265, May 1956. (Atomic Energy Research Establishment, Harwell, England)

The dramatic developments in the application of nuclear fission, especially in view of the possibilities of future generation of electric power in nuclear power stations, make control of the radiological hazards incident to the handling of large quantities of radioactivity extremely important. There is no fundamental difficulty in this control. Review of experience in the British Atomic Energy industry indicates a satisfactory standard of health and an industrial accident rate as low as that of the safest industries, while results of personnel monitoring show that no employee of the U. K. Atomic Energy Authority, in recent years, has had an average weekly exposure rate exceeding the maximum permissible. There have been no instances of any detectable injury arising from external irradiation. The aggregate gonad dose entering the population from the work of the Atomic Energy Authority amounted in 1954 to about 0.1 per cent of that due to natural radioactivity.

Protection against inhalation and ingestion of radioactive materials associated with the processing of spent nuclear fuel is achieved by virtually complete containment of activity during normal operations. When access to contaminated enclosures is necessary, specially designed suits fed with clear air from an air line are used. With highly toxic radioactive materials such as plutonium, operations may be carried out in closed boxes under reduced pressure.

Some monitoring instruments are quite complex and large numbers of them may be necessary, especially in the checking of radioactive substances of long biological half-life in the body, where the total accumulation factor results in a very low permissible level in air.

Testing of individuals for internal contamination is done by measuring, outside the body, the gamma radiation from gamma emitters or the bremsstrahlung from beta-emitting substances. Refined radiochemical technics detect very small amounts of radioactive substances in urine, feces, and blood.

Filtration of air discharged to the atmosphere is carried out to prevent environmental hazard from inhalation or ingestion when this air might contain radioparticulate activity. Safe levels of discharge depend on metabolic properties of the particular element in human beings or in plants and animals which might contribute to human food. Methods are being worked out to retain radioactive gases in the power plant.

Safety in disposal of radioactive wastes presents a problem, especially with the isotopes of relatively long half-life. This can be solved by the location of power plants where waste in the air can cause no harm and other wastes can be stored until no significant radioactivity remains.

Experience so far has shown that radioactivity haz-

ards can be kept under satisfactory control, but only by considerable expenditure of money and manpower. There should be no particular problem in continuing the present satisfactory record into the future nuclear power industry.

Three photographs. RICHARD P. STORRS, M.D.
Los Angeles, Calif.

The Hazards from the Increasing Use of Ionizing Radiations: A Symposium. II. The Dose to Operator and Patient in X-Ray Diagnostic Procedures. G. M. Ardran. Brit. J. Radiol. **29:** 266–269, May 1956. (Nuffield Institute for Medical Research, University of Oxford, Oxford, England)

Until recently, protection of the patient during x-ray examination has been given slight attention, the general view being that, since the patient received the dose only once, it did not matter. Few cases of known injuries to patients and operators have been reported. Most radiologists have appeared to feel that the existing state of affairs is satisfactory.

It has been recognized that leukemia is more common among those exposed to radiation, and geneticists appear to be agreed that any dose of radiation to the gonads will increase the mutation rate and produce effects which are cumulative and probably undesirable.

The recommendations of the International Committee on Radiation Protection (1954) state that "the maximum permissible dose is a dose of ionizing radiation that *in the light of present knowledge* is not expected to cause appreciable bodily injury to a person at any time *during his lifetime.*" In the critical organs, the eyes, blood-forming organs, and gonads, the suggested permissible dose is 0.3 r per week. So far as the gonads are concerned, this figure applies only to those exposed to radiation in the course of their occupation.

The population exposed to diagnostic x-ray procedures as patients is a large and ever-increasing one. In considering genetic aspects, only the population under thirty-five years need be considered, since most people have produced the majority of their children by this age. It is suggested that for large populations the dose should be reduced to 0.03 r per week, and large populations should be considered as not more than one-tenth of the total population.

These recommendations may be interpreted thus: A worker in the radiation field may receive a body or gonad dose of 480 r in thirty years, without noticing ill effects during his lifetime. The patient may on the average receive 48 r to the gonads in the first thirty or thirty-five years if only one-tenth of the population receives this dose.

The available data on the dose received by the patient population from diagnostic procedures are scanty. Estimates of from 0.05 r to 0.2 r per examination have been made.

As a result of added filtration and technical improvements the dose to the patient per examination is less today than it was in the past. It is considered likely that the technological advances have compensated for the increase in number and extent of radiological examinations.

The use of fast film and screens in barium studies of the gastrointestinal tract is recommended to reduce the dose to patient and operators. Image intensification should be used to keep the dosage low during fluoroscopy, especially during examinations on infants and adults still in the reproductive years.

It is important to apply all dose reducing schemes so that the summation of these will result in a significant reduction of the total dose. Radiologists must be aware of the dose levels in their departments and be convinced that they cannot reasonably be lowered. The best way to avoid being forced to chart the patient's dose from cradle to the grave is to find what dose is actually being given to the public and show that it is the irreducible minimum.

Four tables. RICHARD P. STORRS, M.D.
Los Angeles, Calif.

The Hazards from the Increasing Use of Ionizing Radiations: A Symposium. III. An Attempt to Assess the Genetic Changes Resulting from the Irradiation of Human Populations. Alma Howard. Brit. J. Radiol. **29:** 270-273, May 1956. (Mount Vernon Hospital, Northwood, Middlesex, England)

In considering the genetic changes which will be produced by the irradiation of human populations one must consider: (1) the number of mutations produced by a given dose of radiation and (2) the effect of such mutated genes. An upper limit can be set for the number of mutations in relation to dose. The effect of mutated genes of whatever magnitude will be undesirable.

A compilation of studies would indicate that the average dose to the gonads per individual in Great Britain, in millirads per generation (thirty years), is: natural, 3,420; diagnostic examinations, 50; occupational, 3; wrist watches with luminous dials, 60; nuclear detonations, 20.

The genetic structure of a population is determined by the persistence of genes which, in the course of its evolutionary history, were present in individuals best able to survive and reproduce. New kinds of gene types have appeared at random and the direction of forward progress of evolution has been due to selection applied by the environment. With very few exceptions, mutations tend, in a natural environment, to reduce the survival value of the individuals showing them. In man it seems that the spontaneous rate of mutation is already as high as his reproductive activity can tolerate. It is quite probably already higher than optimal, especially since selective forces have undoubtedly altered. When, for example, a child is born to a parent whose life has at any time been saved by medical care, a set of genes persists which would otherwise have been eliminated by natural selection.

In any population, the number of individuals who carry a mutant gene depends upon the rate at which the mutant arises anew and the rate at which it is eliminated before reproduction. A dominant gene which causes death or sterility in every carrier will be eliminated in the first generation and will not accumulate. The majority of mutations, however, have little or no effect on those who inherit them from one parent only. The average persistence of human mutant genes is estimated to be forty generations. The number of accumulated mutant genes of spontaneous origin in the population is estimated to be 8 per individual. Each mutant gives a 1 in 40 chance of early death or sterility. This would indicate that $8 \times 1/40$ of all conceptions end in "genetic death."

Doubling of the spontaneous rate of mutation in one generation would raise the accumulated detrimental mutations to 8.2, an increase of 2.5 per cent. This would mean that one additional person in 200 would

suffer genetic death in the next generation. This is a small proportion and would not be measurable. In absolute numbers, however, it is more impressive, since it means that in Great Britain, with a population of 50,000,000, about 250,000 additional persons in one generation would die prematurely or fail to reproduce as a result of doubling of the spontaneous mutation rate in their parents. In addition, in the generation following exposure, twice the number of congenital defects would be expected. If the spontaneous rate were doubled in each generation for a large number of generations the accumulated results would be much more serious.

An increase of 10 per cent in the natural radiation background for one generation cannot give rise to an increase in mutation rate of more than 10 per cent and it may be nearer 1 per cent, indicating that no more than 25,000 and possibly only 2,500 or fewer individuals in Great Britain would be expected to suffer early death or sterility

While it is true that in attempting to estimate the genetic consequences of radiation we have, at best, only approximate values to use in equations, the qualitative truth of the equations is undeniable. The damage may be described as a risk if we are thinking only of children or grandchildren of an exposed individual or small groups of them, but it is a certainty when considered in terms of future generations of the community and the species as a whole.

There is no "safe" genetic dose. It seems unreasonable to regard any dose as "permissible" unless one implies that one is sure that the advantages resulting from it outweigh the certain damage. The fact that any advantage will be gained by the present generation while the damage will be suffered by later generations makes this a difficult matter to decide. Some compromise is necessary, but it seems very clear that the radiation dose to the gonads should be kept as low as possible.

One table.

RICHARD P. STORRS M.D.
Los Angeles, Calif.

Longevity and Causes of Death from Irradiation in Physicians. Shields Warren. J.A.M.A. **162**: 464–468, Sept. 29, 1956. (194 Pilgrim Road, Boston 15, Mass.)

The author reviews the history of permissible levels of total-body radiation, particularly in America, and cites several studies which have been performed in an attempt to correlate causes of death in radiologists with those in other physicians and the general population. Current reports on the exposure of radiologists, radiographers, dentists, dermatologists, and radioisotope workers are also quoted.

For the present study, the causes of death of physicians and their age at death were taken from the obituary columns of the *Journal of the American Medical Association*, from Jan. 1, 1930, through Dec. 31, 1954. In this twenty-five year period 82,441 deaths were recorded. The age at death was stated for 79,027; the specific cause of death for 65,545. A comparison of mortality of the medical population with the general population twenty-five years or older, showed no difference in mortality rates between the two groups.

In agreement with other studies, the incidence of leukemia in radiologists was found to be approximately one and one-half times that for all specialists in contact with radiation and approximately six times that of physicians assumed not to have been exposed to radiation. The author found that radiologists die, on the average, 5.2 years earlier than do other physicians. The non-radiology specialists known to have some exposure to radiation also show definite shortening of life, but less than radiologists.

It is concluded that exposure to ionizing radiation is the predisposing factor in this shortening of life, and that the degree of precaution against exposure to ionizing radiation employed by some radiologists up to the present time has not been adequate to protect them from its deleterious effects.

This article supports the contention of those who have maintained that any avoidable exposure to radiation is too much. That exposure can be reduced has been shown by the fact that it is possible to operate a general radiology department with an average exposure to personnel of approximately 20 mr per week.

One table.

JAMES E. BAUER, M.D.
University of Missouri

Should We Stop Treating Children with X-Rays? Weighing the Benefits Against Unknown Dangers. Editorial. Antolin Raventos. Pennsylvania M. J. **59**: 1155–1156, September 1956. (3400 Spruce St., Philadelphia, Penna.)

The author cites recent data in the literature implicating radiation therapy in infants as a cause of the later development of thyroid cancer, leukemia, and other malignant conditions. While the evidence that moderate amounts of radiation increase the likelihood of later malignant disease is incriminating, it is not entirely conclusive. It is noted that possible hazards of x-ray treatment have never been adequately investigated by careful long-term follow up.

Comment is made on the recent National Academy of Sciences Report, pointing out that later development of cancer is not the only hazard to be ascribed to irradiation and that gene-transmitted radiation injuries or shortened lives of exposed individuals must also be taken into consideration. The crux of the matter is: how much good can the radiation possibly do as balanced against as yet incompletely understood late dangers. It is concluded that "ionizing radiation is too potent a tool to use in non-malignant diseases except under the most compelling indications." The statement seems particularly true with regard to children.

JAMES W. BARBER, M.D.
Cheyenne, Wyo.

Malignant Disease in Childhood and Diagnostic Irradiation in Utero. Alice Stewart, Josefine Webb, Dawn Giles, and David Hewitt. Lancet **2**: 447, Sept. 1, 1956. (Department of Social Medicine, University of Oxford, Oxford, England)

Public health departments all over Great Britain are engaged in an environmental survey which will eventually cover some 1,500 children who died of leukemia or malignant disease before the age of ten in the years 1953–55. About one-third of the case material has now been gathered, and the authors present an analysis of the findings to date.

One important difference between the children who died and their controls is apparent: The number of mothers who had an x-ray examination of the abdomen during the relevant pregnancy was 85 for the cases and only 45 for the controls. In the group labelled "Other

Past Histories of X-ray Examinations in 547 Children with Malignant Disease and 547 Controls Matched for Age, Sex, and Locality

Period	Type of exposure	No. of Mothers and Children X-Rayed—Leukemia 269 cases	269 controls	Other Malignant Disease 278 cases	278 controls	All Malignant Diseases 547 cases	547 controls
Antenatal	Diagnostic						
	Abdomen	42	24	43	21	85	45
	Other	25	23	33	32	58	55
Before conception of survey child	Therapeutic	—	—	—	1	—	1
	Diagnostic						
	Abdomen	17	24	28	30	45	54
	Other	103	88	108	119	211	207
Postnatal (children only)	Therapeutic	1	—	1	—	2	—
	Diagnostic	45	49	46	50	91	99
	Shoe-fittings	55	52	40	46	95	98
Total no. of mothers		140	130	160	154	300	284
Total no. of children		89	91	75	84	164	175
Either mother or child x-rayed		179	172	194	198	373	370

Malignant Disease," the figures for growths in different parts of the body were:

	Cases	Controls
Brain and appendages	11	9
Kidneys	10	2
Suprarenals	9	4
Lymph nodes	4	2
All other sites	9	4

The authors believe that the findings thus far indicate that, besides producing genetic damage, roentgen pelvimetry may occasionally cause leukemia or cancer in the unborn child.

Diagnosis and Treatment of Radiation Damage to the Gastrointestinal Tract. A. Senn and P. Lundsgaard-Hansen. Schweiz. med. Wchnschr. **86:** 1015–1020, Sept. 15, 1956. (In German) (Chirurgische Klinik der Universität Bern, Bern, Switzerland)

Six to eight weeks after irradiation of the gastrointestinal tract there appears an acute reaction of the intestinal mucosa, with hyperemia and edema and at times transient (spastic?) obstruction, with eventual return to normal. When, however, excessive damage results in deep ulceration, this may lead to gangrene and/or perforation (*i.e.*, peritonitis, fistula, abscess), which leaves as its sequel a chronic stricture, often asymptomatic but sometimes complicated by chronic bleeding and/or mechanical ileus. According to the site of irradiation, the lesions will involve the rectosigmoid (following treatment for carcinoma of the cervix, uterus, ovary), the bowel beyond the reach of the proctoscope (in this group the most frequent cause is irradiation for carcinoma of the cervix), or the stomach (following supervoltage roentgen therapy for seminoma)

Treatment must be individualized. Generally speaking, conservative methods may have a reasonable chance of success in involvement of the rectosigmoid, in view of the fact that surgical removal of the affected segment is not always successful, and often technically difficult. For small bowel lesions, surgery is the method of choice; conservative treatment is seldom helpful. On the contrary, with damage to the stomach, gastrectomy is advisable only for perforation or persistent bleeding, as comparatively good results can be obtained with bland medical treatment.

Four observations are reported: (1) bleeding stenosis of the rectosigmoid after irradiation for carcinoma of the cervix, with good results following resection; (2) partial small bowel obstruction after roentgen, radium, and radiogold therapy for carcinoma of the ovary, treated by surgical removal with end-to-end anastomosis; (3) perforation of the ileum and rectum after irradiation for carcinoma of the cervix, with death six months after surgery, although autopsy failed to demonstrate any residual neoplastic tissue; (4) severe and persistent intestinal bleeding after betatron irradiation following gastric resection for carcinoma of the stomach, not improved after additional (negative) exploratory laparotomy (patient left against medical advice).

E. R. N. GRIGG, M.D.
Cook County Hospital, Chicago

X-Ray Hazards from Television Apparatus and Cathode-Ray Tubes. L. B. Bourne. Lancet **2:** 510, Sept. 8, 1956. (A. C. Cossor Ltd., London, England)

An investigation was made of the radiation received by research workers engaged in developing and testing all types of television and similar equipment which employs cathode-ray tubes operating at higher voltages (up to 20 kv) than those sold for general use. The average dose in 10 workers, who wore monitoring films, was found to be less than 0.05 rad per week, which is below the tolerance level specified by the International Commission on Radiological Protection. It therefore appears that there is little danger to the public from x-rays from television sets working under home conditions if cathode tubes of the present voltage continue to be used.

A Histological and Cytophotometric Study of the Effect of X-Rays on the Mouse Testis. John H. D. Bryan and John W. Gowen. Biol. Bull. **110:** 229–242, June 1956. (Department of Genetics, Iowa State College, Ames, Iowa)

Irradiation-induced changes in reproductive organs

and gametes of various organisms have been the subject of numerous publications and experiments. More recently the emphasis has been placed upon quantitative histological effects of irradiation on the testes. The present paper is a report on a different approach, namely, a combination of a quantitative histologic method together with cytophotometry, giving data with respect to the DNA content of irradiated tissues as well as changes in the frequency of cells. Two inbred strains of mice were used for this study.

With the aid of a cytophotometer it is possible to measure the DNA-Feulgen content of individual spermatogonial nuclei in interphase. Irradiation brings about an inhibition of DNA synthesis, as measured by the uptake of radioactive precursor molecules. Hence, after irradiation, it would be expected that those nuclei which had been prevented from reduplicating their DNA content would be unable to enter mitosis. It is not possible to determine directly whether or not the first spermatogonia to enter mitosis, after irradiation, are those which, though possessing the requisite amount of chromosomal material, had been prevented from dividing. However, the cytophotometric data strongly suggest that this is the case.

The most sensitive stage appears to be that part of the mitotic cycle lasting from late interphase to early prophase. At the time of irradiation, the further away a cell is from this critical stage in its development, the less it is retarded. Similarly, cells which have progressed beyond this stage at the time of irradiation do not undergo inhibition of mitosis. These observations afford a partial explanation for the authors' findings that the rise in the mitotic rate during the period of eight to twenty-four hours after irradiation is followed by a further and more extensive decline.

The suppression of DNA synthesis produced by irradiation appears to be induced in cells which were irradiated in interphase since cytophotometric investigations by various workers indicate that reduplication of the DNA content, in preparation for the next division, takes place during this phase of the mitotic cycle.

The present work shows that the proportion of necrotic spermatogonia constitutes only a minor fraction of the total and supports the alternate hypothesis that irradiation-induced maturation depletion of the seminiferous epithelium is brought about mainly through the irradiation-induced changes responsible for the inhibition of mitotic activity, with cell death playing only a minor role in this process. The work does not support the spermatogonial cell death hypothesis. Instead, the data are consistent with the view that the major effect of irradiation is the suppression of mitosis due to inhibition of DNA synthesis and to the prolongation of prophase through the disturbance of other physiological and biochemical processes necessary for normal mitotic activity.

Four figures; 4 tables.

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Five- to Eight-Day Radiation Death in Mice. Mildred K. Austin, Morton Miller, and Henry Quastler. Radiation Res. **5**: 303–307, September 1956. (Department of Physics, University of Illinois, Urbana, Ill.)

It is claimed that deaths in mice occurring five to eight days after x-irradiation are, as a rule, expressions of a mode of acute radiation death which is different from marrow death and intestinal death. The evidence is threefold:

(1) *Statistical:* Under favorable conditions, the distribution curve of survival time has one or two strong modes between five and eight days.

(2) *Shielding:* Marrow death does not occur if sizable parts of the body are shielded, but death in five to eight days does occur.

(3) *Pathology:* Animals which died at five to eight days showed no denudation of the intestinal walls and regenerated crypts. This excludes intestinal death.

The target zone for this mode of radiation death seems to be in the abdomen.

Two figures. AUTHORS' ABSTRACT

Effect of Total Body Irradiation on Absorption of Sugars from the Small Intestine. John T. Farrar, Melvin D. Small, Doris Bullard, and Franz J. Ingelfinger. Am. J. Physiol. **186**: 549–553, September 1956. (Department of Medicine, Boston University School of Medicine, Boston, Mass.)

A study was made in rats of the effect of large doses (1,410 r) of total-body x-irradiation on intestinal absorption of glucose, fructose, and xylose administered intraduodenally. The irradiation was found to produce a slight but highly significant decrease in absorption of all three sugars when given singly. This investigation does not indicate selective inhibition of any specific mechanism involved in the absorption of these three sugars. No difference attributable to sex existed between the postirradiation absorption rates. When mixtures of the three sugars were given, a questionably significant decrease was observed in all absorption rates, with the exception of xylose in female rats. In this group absorption of xylose was unaffected by irradiation. In rats not exposed to irradiation, the relative absorption rates of the three sugars were markedly different when these were given alone and when given in a mixture.

One figure; 4 tables.

The Role of Transfused Leukocytes in Experimental Bacteremia of Irradiated Rats. J. W. Hollingsworth, Stuart C. Finch, and Paul B. Beeson. J. Lab. & Clin. Med. **48**: 227–236, August 1956. (Department of Internal Medicine, Yale University School of Medicine, New Haven, Conn.)

It has been demonstrated that rats subjected to severe irradiation are unable to remove injected *E. coli* from their blood as efficiently as normal rats (Yale J. Biol. & Med. **28**: 56, 1955. Abst. in Radiology **67**: 479, 1956). This defect in bacterial removal improved significantly after cross-circulation of irradiated with normal rats. The beneficial results of cross-circulation indicate that reticuloendothelial function is not impaired and that improvement results from a transfer of some substance in normal blood into the irradiated rats.

Properdin, a bacterial plasma factor which might logically be implicated in the removal of the *E. coli*, was not found to be of importance. Injection of zymosan, a material obtained from yeast, into normal rats in an attempt to produce properdin deficiency did not adversely affect bacterial removal. An attempt was made to exclude all plasma factors by replacement transfusion of the irradiated rat with heparinized fresh blood, containing everything except leukocytes, from normal rats. In these experiments the buffy coat was simply aspirated and discarded, leaving 500 to 1,700

leukocytes per cubic millimeter. Questionable improvement in the ability of the irradiated rat to remove bacteria was observed, but the most probable explanation for this seemed to be incomplete leukocyte removal from the normal blood. If a plasma factor was involved, the improvement noted after replacement transfusion would have been greater than after cross circulation. It is postulated that the leukopenia of the irradiated rats was responsible for the increased bacteremia and that the transfused leukocytes corrected the defect.

Four figures.

Early Hypotension Induced in the Rabbit by Whole Body X-Irradiation. Phillips M. Brooks, Herbert B. Gerstner, and Sidney A. Smith. Am. J. Physiol. **186:** 532–536, September 1956. (Department of Radiobiology, USAF School of Aviation Medicine, Randolph Field, Texas)

In the dose range between 100 and 1,000 r, whole-body exposure to ionizing radiation produces in mammals, including man, a rather uniform sequence of events: first, an initial reaction occurring within twenty-four hours after irradiation; second, a latent period, with relative well-being; third, a febrile phase associated with severe clinical symptoms; and fourth, either death or prolonged convalescence leading to eventual recovery. Two experiments restricted to the circulatory and respiratory changes occurring during the initial reaction are described.

In the first, the aortic blood pressure and the respiratory chest movements were continuously recorded in 21 rabbits given 630 r total-body x-irradiation at a dose rate of 10.6 r/min., and these recordings were compared with those of 15 control animals. All irradiated rabbits showed a pronounced initial reaction characterized by severe hypotension and acceleration of the respiratory rate. This reaction became noticeable in the second half of the irradiation period; in the majority of animals circulatory collapse with a mean pressure of 50 mm. Hg or less developed during the first hour postirradiation. Thereafter, all rabbits began to recover from the collapse with the exception of 4 which died.

In the second experiment, blood of 16 animals irradiated under the conditions of the first experiment was withdrawn during the collapse phase and the plasma lyophilized. In comparison with control plasma, the lyophilized irradiated material produced a slight but consistent fall in blood pressure when injected into non-irradiated test rabbits.

Four figures; 1 table.

Liver Function in the Chick Following X-Irradiation. Plasma Amino Acids and Plasma Glucose. Elaine Katz Bernstein, S. Phyllis Stearner, and Austin M. Brues, with the technical assistance of Emily J. B. Christian and Betty M. Van Dolah. Am. J. Physiol. **186:** 543–548, September 1956. (Division of Biological and Medical Research, Argonne National Laboratory, Lemont, Ill.)

Abnormal levels of some plasma constituents influenced by the activity of the liver were studied in three-day-old chicks after single dose x-irradiation of 1,000 r, given at 43 r/min. (200 kv, 15 ma, 0.5 mm. Cu and 3.0 mm. Bakelite filtration). An approximately twofold increase in total plasma amino acid concentration was observed at one hour following irradiation, and an approximately threefold increase at three hours. Although the average increase over control values at five hours was the same as at three hours, some individual values increased to about four times the control level. Concurrently, total plasma protein decreased. There was a lack of correlation between the condition of the irradiated animal and the amino acid level or number of amino acids; increased levels occurred at one hour following irradiation in animals which appeared normal at autopsy. A similar alteration in amino acid levels has been reported in man immediately following irradiation; this seems to be an extremely sensitive radiation response.

Mean plasma glucose levels determined in newly hatched chicks at one, three, and five hours after irradiation showed no significant changes from control levels. In individual animals, however, there was a hyperglycemia of 50 to 100 mg. per cent over control values at three and five hours. Such hyperglycemia was usually found in sick birds and was commonly associated with liver hemorrhages.

Three tables.

Investigation of the Lethal Effect of X-Irradiation on the Young Axolotl (Siredon mexicanum). V. V. Brunst. Radiation Res. **5:** 267–280, September 1956. (Laboratory of Radiobiology, Roswell Park Memorial Institute, Buffalo, N. Y.)

One hundred and twenty axolotls from the same spawning were divided into seven groups. The first group was irradiated with 3,000 r. In the second group only the body (exclusive of the head), and in the third only the head was irradiated with the same dose. Groups three, four and five were similarly irradiated, but with 6,000 r. The seventh group constituted an unirradiated control.

All animals irradiated totally with 6,000 r died within thirty days after treatment, and all animals irradiated totally with 3,000 r died within forty days after treatment. In the second group (irradiation of the body exclusive of the head) some animals lived more than 100 days after treatment. After irradiation of the head only, there were some survivals for more than 150 days with 6,000 r and more than 240 days with 3,000 r. Suppression of growth and mortality were greatest after total-body irradiation, smaller after irradiation of the body exclusive of the head, and the smallest after irradiation of the head only.

The primary acute reaction which was described after local irradiation can be observed also after total-body irradiation and after irradiation of the body exclusive of the head, but with great modifications.

A great variety of individual reactions to irradiation were observed, even after total-body irradiation with lethal doses. Even under these conditions complete damage of an entire tissue or organ was noted only in rare cases. (Usually definite damage was observed only in some regions of the tissue, and other regions were normal in appearance.)

The causes of roentgen death after total-body or partial irradiation are different in various animals. In some cases death is seemingly the result of damage to the skin and gills, which are both very important for the normal metabolism of this species. In other cases it is the result of damage to the intestinal tract, connective tissues, spleen, kidney, or liver.

AUTHOR'S ABSTRACT

Lethality Induced by Feeding Radiophosphorus to Male Habrobracon. Daniel S. Grosch. American Naturalist **90:** 200–202, May-June 1956. (Department of Genetics, North Carolina State College, Raleigh, N. C.)

Dominant lethality may be induced experimentally in the offspring of male Habrobracon fed P^{32}. However, this type of experiment is difficult to set up with wasps because the males do not withstand the preliminary starvation as well as females. Furthermore, radioactively equivalent feedings are not as biologically effective as in females. In contrast with the ovaries, the testes show no concentration of radioisotope when studied by autoradiography.

One table. AUTHOR'S ABSTRACT

The Distribution of Energy Imparted to a Homogeneous Medium Non-uniformly Irradiated. G. Failla. Radiation Res. **5:** 205-215, September 1956. (Radiation Research Laboratory, 630 W. 168th St., New York 32, N. Y.)

The flux, dose rate, and other characteristics of the primary and secondary radiations in non-uniform radiation fields are described and discussed. It is shown that, while uniform overall changes in the density of the medium result only in a "photographic" enlargement or reduction of the flux pattern, in which the proper geometric relationships are preserved, local density changes produce serious distortions. It is for this reason that a uniform radiation field is generally stipulated for measurements based on the Bragg-Gray principle. In a non-uniform field the cavity, even if filled with a gas of the same atomic composition as the wall, introduces flux disturbances. The geometric effect of the cavity *per se* may, however, be eliminated by use of an extrapolation type of ionization chamber. The principles discussed in the paper are useful in this connection and in the design of ionization chambers for the measurement of absorbed dose under unfavorable conditions. They are also useful in arranging the exposure conditions in radiobiological experiments so that more accurate determinations of absorbed dose can be achieved.

Four figures. AUTHOR'S ABSTRACT